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Segmental Arterial Spasm in Patients with Total Brain Infarction

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ABSTRACT

An arteriographic investigation has shown that segmental spasm occurs in a relatively high frequency of patients with total brain infarction (12 of 30), and if spasm at the origin of arterial branches is included, the frequency is still higher (19 of 30). The phenomenon is possibly a sign of changed sympathetic tone, the pathophysiological significance of which is discussed.

INTRODUCTION

During the last 20 years sporadic reports have been made of wave-like, regular, inconstant luminal variations in arteries at arteriography. This phenomenon is usually limited to one segment of the artery and arteriographically it somewhat resembles fibromuscular hyperplasia. It differs from the latter condition, however, by the softness and strict regularity of the waves. Furthermore, these waves can be altered or made to disappear completely by another injection of contrast medium, with or without previous injection of a vasodilative agent. The length of the wave in the affected segment is correlated to the diameter of the artery. This arteriographic phenomenon will be referred to in the following as a segmental spasm (SS).

The first to report this observation was Ratschow (13). He believed it to be due to a dysfunction of the vessel, and claimed that occurrence of the phenomenon on the arteriogram was related to pain. Wickbom & Bartley (18) found that arterial "spasm" were eliminated by Priscol[1] and claimed that a proneness to spasm was more marked in patients with Raynaud's disease. They also pointed out that the frequency of the phenomenon was inversely proportional to the age of the patient, which they attributed to increasing rigidity of the vessel wall with advancing age. Lindbom (10) reported that spasms could be induced by raising the intravascular pressure or by stretching the artery. The term "standing waves" was used by Theander (16), who concluded from measurements on films that the length of the waves in the involved segment was directly related to the diameter of the artery, and also observed that in one case the vessel was wider when stationary waves were noted than when they were absent. From these findings and from physical arguments he considered that the phenomenon could not be due to spasm but was caused by resonance in the vessel proximal to an occlusion. This theory was questioned by Köhler (9) who made a physico-physiological analysis of the standing wave phenomenon and demonstrated that the arteriographic length of the assumed standing wave in human arteries did not correspond to the luminal variations.

Köhler considered that the changes were probably caused by a spastic circular constriction of the arterial wall, and were very likely an artificial phenomenon produced by an irritation from the contrast medium in an especially sensitive vessel. Mayall (11) postulated that the wavy contour was due to the formation of layers between the blood and the contrast medium, with rippling at the interface between these layers. This theory was refuted, among other authors, by New (12), who pointed out that the phenomenon could also be perceived with a horizontal roentgen beam. Ishikawa et al. (6, 7) reported a series of arteriographies of the lower extremity, in which accordion-like arterial shadows were present in about 10%. They claimed that this high frequency was related to the fact that the examination was performed with semiflexed legs and under spinal anesthesia, conditions which

[1] A substance with a moderately effective adrenergic blocking function.

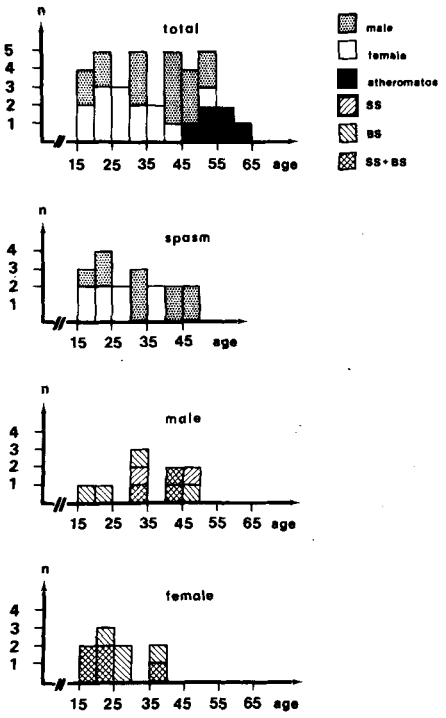

Fig. 1. Age distribution in the total material and in the patients with spasm. The type of spasm with regard to patients age and sex.

seemed to predispose to longitudinal contraction of the artery. They also pointed out that the phenomenon persisted after manual compression of the artery proximal to the site of injection. On electrical stimulation of the sympathetic nerves supplying the kidney in the rabbit, Bergquist et al. (2) found luminal variations in the renal artery reminiscent of those observed in clinical segmental spasm. Segmental spasm has been reported from practically all arterial areas except intracranial and coronary arteries—vital vascular areas in which an arterial spasm would endanger the patient's life. Segmental spasm has not been reported in patients examined under general anaesthesia.

The causative mechanism of the phenomenon has been much discussed but little attention has been paid to its pathologic significance and its possible relation to the patient's symptoms. Its occurrence has been reported in Raynaud's disease (18), Bürger's disease (15) and angiodyskinesia (1, 5, 8). No uniform group of patients has been presented, however.

In recent years some reports of nonocclusive ischemia in the small intestine in association with cardiovascular shock have been published (19, 20). Siegelmann et al. (14) demonstrated spasm of the superior mesenteric artery in the dog in various types of cardiovascular shock in angiographic investigations; this spasm was located at the origin of arterial branches, and caliber variations were found in the main trunk or branches of the superior mesenteric artery.

We have observed segmental spasm at some arteriographies in our clinic. A relatively large number of these cases have been patients with total brain infarction. For this reason we have reexamined a series of arteriograms from patients with this conditions.

MATERIAL AND METHODS

(1) A retrospective investigation was performed on abdominal arteriograms from 36 patients with clinical signs of total brain infarction (areflexia, hypothermia, persistent respiratory arrest as tested by a 3-min apnoea test; isoelectric EEG on two occasions in each patient). On these patients cerebral 4 vessel-angiography was performed to establish the diagnosis and renal aortography in order to survey the renal circulation with a view to pos-

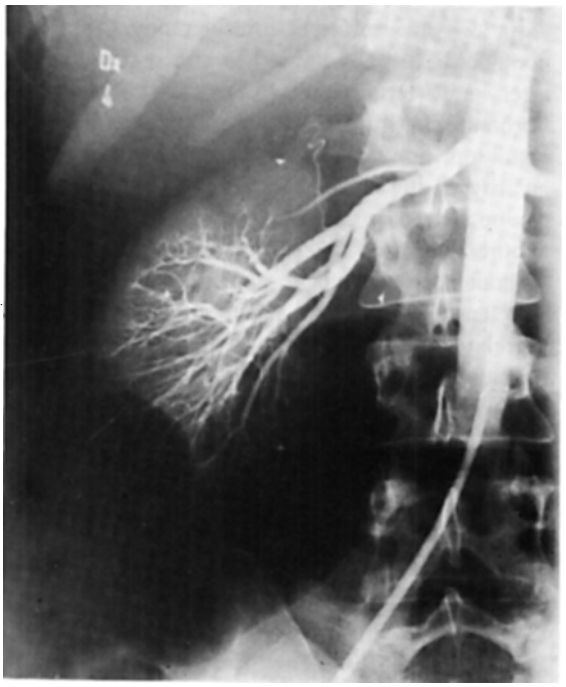

Fig. 2. Patient 14, a woman aged 38 years. Total cerebral infarction following intoxication. A semi-selective arteriogram clearly shows segmental spasm in the right renal artery. The patient was having an isoprenaline drip infusion at the time of the examination.

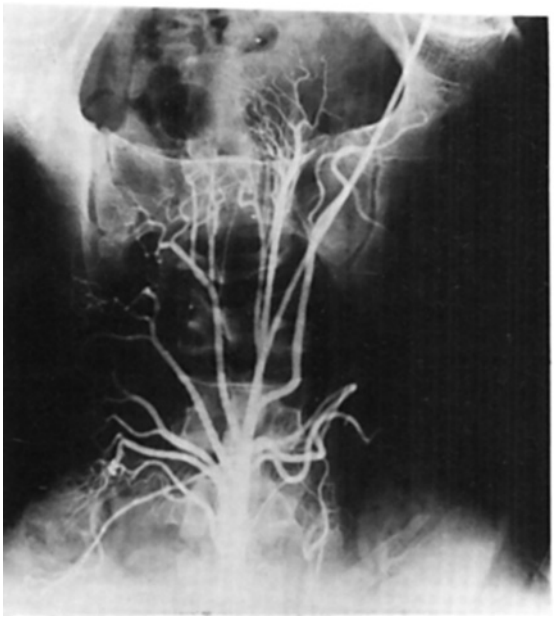

Fig. 3. Patient 21, a man aged 42 years. Total cerebral infarction following an operation for subdural hematoma. Selective angiography of the superior mesenteric artery. No drugs were given during the examination. Note that in addition to segmental spasm there is a distinct spasm at the origin of arterial branches.

sible kidney donation. Six patients with atherosclerosis were excluded. Of the remaining 30 patients, 14 were women and 16 men. The age and sex distributions are shown in Fig. 1.

(2) Clinical data were obtained from the case journals. The pulse, blood pressure and temperature were recorded regularly.

(3) Clinico-chemical blood analysis of PO_2, pH and PCO_2, using a blood gas analyzer at 37°C (Instrumentation Lab. Incorp., no. 313), were performed within 24 hours following the roentgen examination. HCO_3 was measured at 37°C with a microcapillary technique on a radiometer pH meter 22 (Radiometer, Copenhagen). Na^+ and K^+ were determined by flame photometry (IL 343 Flame Photometer, Instrumentation Lab. Inc., Lexington, Mass., USA). These data were also obtained from the case journals.

Cerebral 4 vessel-arteriography was performed by the Seldinger technique, using a grey Ödman-Ledin catheter with side-holes, introduced through the femoral artery. The tip of the catheter lay in the ascending aorta.

At renal aortography 30 ml Angiografin was injected at the level of the origins of the renal arteries, i.e. with the catheter tip located at L 1. All injections were given at a pressure of 5 kp per cm^2, using a Cisal II pressure syringe. The exposure frequency during the renal aortography was 3 frames per sec for 3 sec, followed by 1 frame every other sec for 8 sec. At selective arteriography of the superior mesenteric artery 25 ml Angiografin was injected at a pressure of 3 kp per cm^2; the exposure frequency was 2 frames per sec for 5 sec and 1 frame every other sec for 10 sec. This selective arteriography was performed in patients 21 to 30, with the exception of patient 27. At all arteriographies the ECG, injection time and exposure were registered with a Mingograf En 81 or Mingograf 800 direct recorder. The film–focus distance was 100 cm in all examinations and the same apparatus was used on all patients included in this investigation. The exposure data were 60–75 kV, 500–600 mA and 0, 10 sec.

At the re-examination of the abdominal arteriograms, the renal and superior mesenteric arteries were examined with respect to segmental spasm and the latter arteries were also examined for spasm at the origins of branches. Spasm at the origins of branches was considered to be present if at least two of the branches were slightly but clearly narrowed for a very short distance (1–2 mm).

RESULTS

(1) All examined patients showed clinical signs of total brain infarction, and this was also evident at cerebral 4 vessel-angiography.

(2) The systolic blood pressure was low in all cases (50–100 mmHg). The rectal temperature was also low and varied between 31 and 35.2°C, with the exception of one patient (no. 16), whose temperature was 39°C. This latter measurement was possible wrong. The heart rates varied from 50 to 150 beats per min at the time of the angiographies. See Tables I and II.

(3) The PO_2, PCO_2, pH and serum electrolyte values are given in Tables I and II. At the time of the angiographies the patients were being treated with the drugs listed in Table I.

(4) Of the 30 patients, one had segmental spasm in the right renal artery. Eleven had segmental spasm in the superior mesenteric artery or its branches. Sixteen patients had spasm at the origin of two or more branches of the superior mesenteric artery. Nine patients had both segmental spasm and spasm at the origin of arterial branches. (See Tables I and II). Only in 11 patients was no form of spasm observed.

DISCUSSION

In 6 patients atheromatosis was noted. These patients were relatively old and none of them showed signs of spasm, possibly because of rigidity of the vessel wall. They were therefore excluded from the subsequent analysis. The reason for the high frequency of spasm in patients with total brain infarction can be assumed to be associated with the clini-

Table I. *Data for the individual patients: age, sex, diagnosis, drugs given, blood pressure, heart rate, temperature and clinico-chemical laboratory values*

Unfortunately the case journal for patient 1 could not be found. S=segmental spasm; BS=spasm at origin of branches.
Drugs: a, atropine; b, insulin; c, isoprenalin; d, antibiotics; e, phenytoin; f, furosemide; g, corticosteroids; h, chlorpromazine

No.	Sex	Age (yrs)	SS	BS	Diagnosis	Drugs	Syst. BP (mmHg)	HR (beats/ min)	PO_2 (mmHg)	PCO_2 (mmHg)	HCO_3^- (mmol/l)	pH	Na^+ (mmol/l)	K^+ (mmol/l)	Temp. °C
1	♂	52	–	–	ic hemorr										
2	♀	20	+	+	subdur hemat	d, g	70	50	–	22.5	20	7.5	134	3.1	31.0
3	♂	40	+	+	fract skull	g	90	70	172	33	24	7.46	143	5.7	34.2
4	♀	23	+	+	fract skull	d, g	90	70	159	35	28	7.45	144	4.0	34.5
5	♀	16	+	+	fract skull	d, g	70	60	236	21	14	7.34	137	3.9	32.8
6	♀	34	–	–	subarach	f	70	60	141	19	22	7.55	138	3.9	33.0
7	♀	15	+	+	subarach	d	70	80	–	21.5	21.5	7.52	151	4.3	34.8
8	♂	30	+	+	subarach	–	90	80	256	34	16	7.43	143	3.7	35.0
9	♂	23	–	–	subarach	h, c	100	150	108	42	20	7.27	143	4.0	34.9
10	♂	52	–	–	fract skull	f	90	80	70	37	17	7.38	139	2.5	33.7
11	♂	46	–	–	op tc	f, h	50	90	–	–	–	–	135	5.1	35.2
12	♀	53	–	–	subarach	f, h	60	70	78	30	13.5	7.4	129	3.7	34.2
13	♀	30	–	–	subarach	f, h	70	80	–	–	–	–	130	2.2	33.2
14	♀	38	+	–	intox barb ethyl	c	100	50	98	19	12	7.37	150	2.9	32.6
15	♂	18	–	–	fract skull	f, g	70	120	165	57	23	7.26	148	3.9	35.2
16	♀	29	–	–	op ic aneur	e, f, g, h	100	100	119	30	24	7.47	132	3.9	39.0
17	♂	41	+	+	subdural hemorr	e, f, g, h	80	90	99	41	28	7.44	147	3.6	33.4
18	♀	28	–	+	fract skull	g, h	70	70	103	18	21.5	7.7	148	3.1	32.5
19	♂	41	–	–	op ic hemat	–	60	70	–	–	–	–	133	6.1	33.0
20	♂	23	–	+	fract skull	e, g	90	80	164	49	27.5	7.21	143	3.3	34.0
21	♂	42	+	+	op subd hemat	–	80	100	107	34	33	7.58	146	3.7	35.0
22	♂	45	+	–	cer hemorr	g	90	60	–	–	17.5	–	146	4.2	32.0
23	♀	35	+	+	subarach	a, d	110	80	102	33	23.5	7.51	139	4.0	31.5
24	♀	40	–	–	subarach	–	90	70	–	–	23.5	–	158	4.1	35.0
25	♀	22	–	+	fract skull	f	70	60	–	–	25.5	–	158	4.1	31.0
26	♂	19	–	+	op aneur	c, g	100	80	126	45	22	7.36	165	2.4	34.5
27	♂	32	+	–	fract skull	b	90	70	–	–	26.0	–	153	1.8	31.5
28	♂	49	–	+	fract skull	c	80	130	–	–	26.0	–	145	4.4	33.0
29	♂	30	–	+	fract skull	d, g	90	70	67	34	24.5	7.6	154	4.4	33.2
30	♀	25	–	+	encephalitis	c, d, g	80	80	–	–	26.0	–	166	4.0	34.0

Table II. *Shows the statistical datas for the different groups of findings with respect to arterial blood pressure, heart rate, arterial PO_2, PCO_2, HCO_3^-, pH, serum Na^+, K^+ and body temperature*

	BP (mmHg)	HR (beats/min)	PO_2 (mmHg)	PCO_2 (mmHg)	HCO_3^- (mmol/l)	pH	Na^+ (mmol/l)	K^+ (mmol/l)	Temp. (°C)
SS									
M	86	71	153	29.4	21.9	7.46	144	3.7	33.2
n	12	12	8	10	12	10	12	12	12
S.D.	12	15	64	7.6	6.3	0.07	6	0.9	1.5
Range	70–110	50–100	98–256	19.0–49.0	12.0–33.0	7.34–7.58	134–153	1.8–5.7	31.0–35.0
S at origin									
M	83	81	115	36.5	24.7	7.47	155	3.7	33.2
n	7	7	4	4	7	4	7	7	7
S.D.	11	22	41	13.9	2.2	0.22	11	0.7	1.2
Range	70–100	60–130	67–164	18.0–49.0	21.5–26.0	7.21–7.70	143–172	2.4–4.4	31.0–34.5
All spasm									
M	85	75	141	31.4	23.0	7.46	148	3.7	33.2
n	19	19	12	14	19	14	19	19	19
S.D.	12	18	58	9.8	5.3	0.12	9	0.8	1.3
Range	70–110	50–130	67–256	18.0–49.0	12.0–33.0	7.21–7.70	134–172	1.8–5.7	31.0–35.0
No spasm									
M	76	86	113	35.8	20.4	7.38	138	3.9	34.6
n	10	10	6	6	7	6	10	10	10
S.D.	18	28	36	12.9	3.9	0.11	9	1.1	1.8
Range	50–100	60–120	70–165	19.0–57.0	13.5–24.0	7.26–7.55	129–158	2.2–6.1	31.5–39.0

cal status. These patients comprise an extreme group—their higher brain functions have been eliminated and their respiratory centre is out of function. Their blood pressure and body temperature are low, and the heart rate is also usually low. The electrolyte balance is frequently disturbed and artificial ventilation is necessary, entailing a risk of changes in the acid-base status. This retrospective investigation revealed no difference between patients with and those without spasm with respect to blood pressure, temperature, blood gases, blood pH and serum sodium and potassium. Four of five patients treated with an isoprenaline drip infusion, 11 of 13 patients treated with cortisone, and all 6 patients receiving antibiotics had segmental spasm. The material is too small, however to draw any conclusions from these observations.

It is interesting that there appear to be no reports on segmental spasm in children, whereas adolescents are well represented in various series. One possible explanation for this is that in children arteriographies are performed under general anaesthesia, which affects the vascular tone. Preliminary results of direct nerve recordings in man show that the sympathetic activity in cutaneous nerves is inhibited on induction of anaesthesia (17) with fluothane. A similar anaesthesia might be the reason for the absence of segmental spasm in children. The sympathetic activity is altered if the impulses from higher centers to the vasomotor center are eliminated (3, 4). It is thus possible that the sympathico-adrenal activity in patients with total brain infarction has a completely different profile from that in other individuals; this is possibly to be regarded as dysfunction of the sympathetic nervous system. The simultaneous occurrence of spasm at the origins of branches of the superior mesenteric artery and segmental spasm in the same vascular area supports the theory that segmental spasm is caused by a change in tone of the arterial wall. The high frequency of spasm in a group of patients with total brain infarction may thus mean that segmental spasm is related to changes in the sympathetic activity of the individual. Applied to clinical routine, the occurrence of segmental spasm may thus, in certain circumstances, be a sign of altered vascular tone, the late effects of which are relatively uninvestigated. Thus the phenomenon may imply a change (possibly an increase) of the sympathetic tone of the patient. It would be of interest

to investigate whether in healthy persons this phenomenon is a manifestation of a sympathetic dysfunction, which could cause changes in the regional blood flow.

Segmental spasm should not be confused with fibromuscular hyperplasia. If any doubt should arise as to the nature of the arterial changes, it is recommended that a vasodilatant e.g. Bradykinin be injected into the artery via the catheter inserted for the injection of contrast medium. A spasm might then be expected to change or disappear, while waves caused by an organic mechanism would be unaffected.

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