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Kinking of the human internal carotid artery: a statistical study in 100 healthy subjects by echocolor Doppler

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Background. **Kinking of the internal carotid artery is a rather frequent anomaly. It is still controversial whether it has a clinical relevance, causing cerebral vascular disturbances, or if it is a mere morphologic anomaly. A study concerning its frequency in asymptomatic subjects and an evaluation of its possible correlations were, therefore, considered interesting.**
Methods. **The internal carotid artery was studied in 100 apparently healthy subjects (50 men and 50 women; age range: 18 to 98 years) with no history of atherosclerotic disease of the supra-aortic trunks, or diabetes mellitus, or hypertension. Each patient underwent color Doppler ultrasonography.**
Results. **In 38 subjects out of 100, and in 53 out of 200 examined arteries (100 right and 100 left arteries), a kinking was found (26.5%). In 23 kinkings (43% of all the kinkings), the maximal systolic velocity (MSV) was greater than 1 m/sec at the level of the kinking. The kinkings were more frequent in women; such prevalence was statistically significant. The frequency of kinkings was uncorrelated with the side of the artery, the age, the body weight, the height and the length of the neck. Moreover, the presence of the kinkings was not significantly correlated with the lumen of the internal carotid artery.**
Conclusions. **Hypotheses regarding the genesis of the kinkings were discussed, and the embryogenic hypothesis was emphasized. The importance of the MSV in the diagnosis of kinkings was stressed.**

KEY WORDS: Carotid artery, internal abnormalities - Carotid artery diseases ultrasonography.

The study of the morphologic variants of the internal carotid artery (ICA) along its extracranial course has an acute interest in consideration of possible clinical involvement.[1-4]

As already noticed by Morgagni in 1700, the convoluted enlargements are particularly interesting because of their frequency.[5] In such cases, the ICA can show different aspects, as coiling, kinking or looping. In the kinking, the most frequent pattern,[6 7] two segments of the artery form an acute angle.[8] Traditional anatomic studies give only few notices about this vascular behaviour [9-11] and extensive investigations concerning the kinkings were performed many years ago.[12] On the other hand several studies, both surgical [5 7 13 14] and radiologic, [1 6] report this morphological condition, even in asymptomatic patients.

Up to date, the onset of more and more sophisticated diagnostic imaging techniques could allow a better evaluation of such apparent disagreement. Ultrasonography appears to be the most suitable technique: it is the simplest non-invasive method, and allows wide-range studies of cardiovascular anatomy in healthy volunteers, taking minimal disturbances for patients.[15] The ultrasonographic investigations using high-definition echotomography, enhanced with a Doppler module and color codification, allows accurate real-time measurements of vessel lumen and blood flow, in addition to a qualitative assessment of blood flow.[16-18]

The aim of the present study is to evaluate the incidence of carotid kinking by an ultrasonographic

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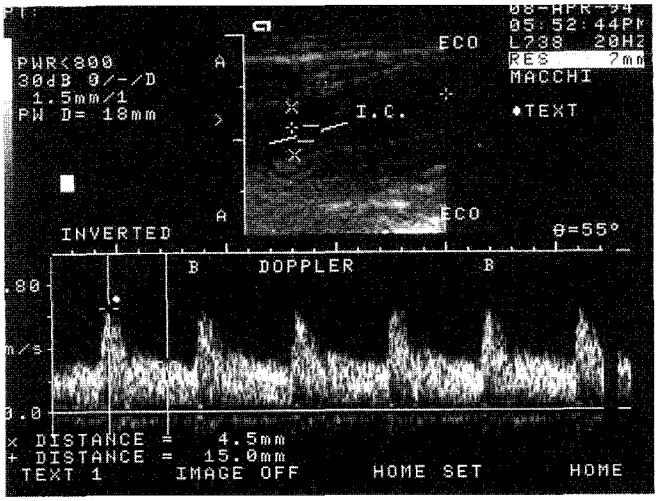

Fig.1.—Echo Doppler illustrating the method for measurement of diameter (1.5 cm after their origin) and systolic velocity of the internal carotid artery.

study of a wide population of patients asymptomatic for carotid diseases.

Materials and methods

One hundred healthy volunteers (50 men, 50 women; age range: 18 to 98 years), with no history of atherosclerotic disease of the supra-aortic trunks, diabetes mellitus, or hypertension, were considered. The patients referred no clinical symptoms of defective carotid flow. All subjects gave their informed consent to participate in this research.

For each subject, sex, age, height and weight were registered; then the patients underwent a color Doppler examination of the supra-aortic trunks (arteries examined: brachioradialis, subclavian; common carotid; internal carotid; external carotid; vertebral). The following parameters were noted:

1) the lumen of the internal carotid artery (0.5-1 cm from its origin) and, using a pulsed Doppler module, the maximal systolic velocity (MSV) of blood flow at this level (Fig. 1);

2) the morphology of the kinking (if present) and its distance from the vessel origin. When several kinkings were present in the same vessel, the kinking closest to the vessel origin was considered for this measurement;

3) the maximal systolic velocity (MSV) (in meters/second) at the level of the kinking. When several kinkings were present in the same vessel, the measurement was performed in the kinking with the highest velocity;

4) the maximal systolic velocity 0.5 cm distal to the kinking. When several kinkings were present in the same vessel, the distal kinking was considered;

5) the MSV (using a continuous Doppler module), in the terminal branch of the ophthalmic artery with the highest velocity. This measurement was performed in order to evaluate the hemodynamic effects of the kinking, down to a terminal branch of the carotid artery;

6) the length of the neck, measured as the distance between the superior margin of the clavicle and the mandibular angle (measured on both sides and averaged, to avoid possible errors related to tilting of the head with respect to the median plane). This parameter was obtained in supine patients with the head in maximal extension.

The data obtained were processed statistically.

For color Doppler ultrasonography, an Acuson 128 XP apparatus equipped with a 5 MHz probe was used. A thermic printer (Sony UP-850 and Color Video Printer UP-3030P) was used for photographic documentation.

Results

The data obtained are reported in Table I. The mean age of the subjects was 67.5 years (men, 67.8; women, 67.2). Carotid kinking was found in 38 subjects: it was bilateral in 15 cases (9 women, 6 men; 39% of the cases in which kinking was present); left-sided in 12 (8 women, 4 men; 32% of the cases in which kinking was present); right-sided in 11 (7 women, 4 men; 29% of the cases in which kinking was present). Therefore, if one considers all the 200 carotid arteries examined (i.e. 100 right and 100 left vessels together), there was a total of 53 kinkings (Table II). The mean MSV (ranging from 0.45 to 0.65 m/sec in normal internal carotid artery; mean value 0.51±0.14 m/sec) at the level of the kinkings was 0.81±0.10 m/sec for those arteries whose velocities were lower than 1 m/ sec; it was 1.19±0.10 m/sec for those vessels whose velocities ranged from 1 to 1.5 m/sec, and 1.76±0.15 m/sec for those arteries whose velocities ranged from 1.5 to 2 m/sec (Table III).

TABLE I.—*Totality of cases.*

N	S	A	W	H	I.C.d		KK or		S.V.		V.B.		V.A.		LN
					r	l	r	l	r	l	r	l	r	l	
1	F	18	55	1.76	4.20	4.20			0.62	0.60					8.3
2	F	19	54	1.63	5.80	5.50			0.47	0.45					8.7
3	F	28	54	1.63	3.50	3.50			0.86	0.79					11.2
4	F	48	52	1.60	3.40	3.10			0.73	0.68					11.5
5	F	48	65	1.64	5.90	5.70			0.52	0.54					12.3
6	F	49	89	1.77	5.20	5.00			0.46	0.48					10.5
7	F	49	57	1.60	4.60	5.00			0.72	0.74					9.7
8	F	54	78	1.68	4.50	4.70			0.58	0.54					11.3
9	F	56	79	1.74	5.30	4.30	29.40	32.10	0.82	0.93	0.44	0.46	0.41	0.51	12.2
10	F	60	65	1.51	3.30	4.10	44.30	22.00	0.83	0.97	0.52	0.48	0.54	0.53	8.1
11	F	64	63	1.65	4.30	4.30			0.61	0.59					9.3
12	F	64	60	1.50	4.00	4.00		33.90	0.64	1.17		0.72		0.66	7.9
13	F	64	68	1.58	4.30	4.40	38.10		1.77	0.55	0.68		0.94		10.8
14	F	65	68	1.69	4.80	4.90			0.53	0.64					10.2
15	F	65	64	1.58	4.80	4.60			0.67	0.61					10.2
16	F	66	56	1.63	5.20	5.30		4.60	0.51	1.12		0.70		0.51	10.4
17	F	66	75	1.68	3.90	4.00		33.60	0.46	0.86		0.54		0.61	11.2
18	F	66	82	1.68	4.00	4.60		20.20	0.55	1.40		0.46		0.63	12
19	F	67	66	1.60	4.80	4.60	33.60	24.00	0.89	0.90	0.47	0.63	0.69	0.78	7.8
20	F	68	68	1.65	4.70	4.70	28.20	40.10	0.77	1.34	0.55	0.59	0.58	0.77	12.9
21	F	68	70	1.62	4.90	5.20	25.20		0.88	0.49	0.64		0.69		8.9
22	F	68	80	1.66	4.80	4.60			0.58	0.59					9.9
23	F	69	69	1.65	4.10	4.70		33.30	0.79	1.15		0.65		0.50	11.5
24	F	69	67	1.54	5.60	5.40	24.20		1.58	0.53	0.72		0.97		12.2
25	F	70	74	1.76	4.60	4.40			0.60	0.68					11.3
26	F	70	54	1.56	4.50	4.30	16.20	18.00	0.85	1.07	0.56	0.53	0.68	0.79	8.4
27	F	71	54	1.52	4.30	4.50	32.80		0.99	0.56	0.43		0.52		9.7
28	F	72	73	1.68	4.80	4.80			0.49	0.49					9.8
29	F	72	59	1.70	5.50	5.30	3.10		0.82	0.62	0.42		0.56		10
30	F	72	58	1.55	6.10	6.00			0.40	0.39					8
31	F	73	76	1.54	5.00	4.80	19.70		1.35	0.55	0.68		0.74		13
32	F	73	51	1.69	4.20	4.30			0.49	0.55					10.7
33	F	73	61	1.67	4.50	4.30		35.50	0.43	1.90		0.51		0.64	10.5
34	F	73	77	1.68	3.90	4.20			0.49	0.58					10.2
35	F	73	65	1.60	4.20	4.00			0.59	0.52					9.4
36	F	74	75	1.63	5.40	5.20			0.47	0.58					9.5
37	F	74	84	1.64	4.40	4.10			0.53	0.48					9.7
38	F	76	70	1.59	5.10	4.90		24.30	0.58	0.80		0.56		0.56	10
39	F	76	60	1.65	5.10	4.50	24.80	15.80	0.80	1.92	0.54	0.52	0.49	0.99	11.5
40	F	76	74	1.71	5.20	5.00			0.48	0.48					12.1
41	F	77	64	1.52	4.80	4.80			0.44	0.44					8.9
42	F	78	72	1.65	5.90	5.70			0.54	0.53					10.9
43	F	79	61	1.59	5.20	5.00			0.47	0.53					9.1
44	F	80	53	1.64	4.60	4.40	23.60	20.20	0.77	0.57	0.50	0.50	0.58	0.68	9.9
45	F	81	50	1.57	4.70	4.70	36.00		1.65	0.63	0.67		0.72		9.4
46	F	81	49	1.53	4.90	5.10			0.46	0.45					11.2
47	F	84	60	1.64	4.30	4.00	26.60	15.40	1.02	1.12	0.52	0.37	0.52	0.56	10.6
48	F	87	48	1.53	4.60	4.50		17.60	0.49	0.98		0.62		0.79	9.3
49	F	90	62	1.65	4.20	4.30			0.60	0.59					12
50	F	98	68	1.58	4.70	4.80	27.40	28.80	0.92	0.83	0.51	0.51	0.63	0.51	10

N: number of subjects; S: sex; A: age; W: weight; H: height; I.C.d: Internal Carotid lumen at 2.5 cm after its origin; KK or: distance of the kinking from the origin of Internal Carotid Artery; S.V.: maximal systolic velocity at level of the kinking; V.B.: maximal systolic velocity before the kinking; V.A.: maximal systolic velocity after the kinking; r= right; l= left; LN=lenght neck.

Continued Table I

Continued Table I.

N	S	A	W	H	I.C.d		KK or		S.V.		V.B.		V.A.		LN
					r	l	r	l	r	l	r	l	r	l	
51	M	49	59	1.59	4.20	4.20			0.54	0.58					11.6
52	M	50	90	1.72	5.00	4.10			0.71	0.84					8.5
53	M	54	80	1.73	5.10	3.60	30.30	33.00	1.28	1.15	0.54	0.65	0.66	0.64	11
54	M	54	78	1.66	3.80	4.10			0.63	0.70					11.1
55	M	55	73	1.74	4.20	4.00			0.48	0.52					9.8
56	M	55	84	1.79	4.70	4.50			0.58	0.59					11.2
57	M	56	75	1.80	4.70	4.50			0.62	0.63					14
58	M	58	75	1.64	5.20	4.90			0.49	0.60					11
59	M	59	72	1.69	4.40	4.30			0.58	0.58					9.7
60	M	59	78	1.69	4.70	4.90	20.10		0.58	0.52	0.33		0.38		10.5
61	M	59	88	1.72	5.30	4.80			0.46	0.47					12
62	M	62	98	1.77	4.30	4.60	34.60	24.90	1.20	1.28	0.50	0.51	0.60	0.70	8.3
63	M	62	70	1.75	4.30	4.40			0.60	0.64					11
64	M	64	58	1.58	4.00	3.90			0.58	0.59					12
65	M	64	90	1.78	4.10	4.50			0.75	0.67					9.4
66	M	64	##	1.90	4.70	5.30			0.43	0.48					9.3
67	M	65	73	1.67	4.90	5.20		22.20	0.49	0.81		0.58		0.63	8.2
68	M	65	58	1.58	4.60	4.80			0.65	0.64					10.3
69	M	65	65	1.72	4.70	4.30			0.52	0.54					12.3
70	M	66	89	1.78	5.20	5.30			0.63	0.61					9.4
71	M	66	83	1.70	4.50	4.30	20.10	22.30	0.68	0.64	0.55	0.56	0.57	0.54	13
72	M	66	82	1.79	4.80	4.80	22.40	22.60	0.82	0.90	0.49	0.53	0.53	0.58	11.5
73	M	67	87	1.83	5.80	5.40			0.48	0.49					11.7
74	M	68	80	1.70	4.40	4.60			0.59	0.44					8.9
75	M	68	70	1.70	5.50	5.60			0.52	0.49					8.1
76	M	70	79	1.72	4.50	4.50		32.40	0.79	1.09		0.50		0.65	9
77	M	70	65	1.60	4.30	4.10	27.20	24.40	0.76	0.88	0.58	0.68	0.68	0.69	11.8
78	M	70	73	1.77	5.20	5.50			0.53	0.62					10.9
79	M	71	69	1.70	4.60	4.40		40.10	0.53	0.67		0.53		0.65	12
80	M	71	83	1.75	3.90	4.00	20.60		0.68	0.69	0.48		0.63		9.8
81	M	72	70	1.81	5.20	5.00			0.55	0.58					12
82	M	72	65	1.60	4.90	4.80			0.63	0.66					9.2
83	M	72	74	1.72	4.60	4.40	25.60		1.12	0.62	0.47		0.67		10.2
84	M	73	78	1.65	3.90	5.00			0.72	0.62					10.5
85	M	73	63	1.62	5.20	4.90			0.47	0.47					11.8
86	M	73	80	1.70	6.00	6.10			0.46	0.49					8.9
87	M	73	65	1.67	4.80	4.70			0.59	0.47					10.2
88	M	73	77	1.75	4.60	4.60	31.40		1.08	0.58	0.60		0.70		8.8
89	M	74	71	1.76	4.20	4.40			0.49	0.68					9.9
90	M	76	68	1.80	5.50	5.30			0.48	0.47					9.6
91	M	76	66	1.58	5.50	5.10			0.57	0.52					9.5
92	M	77	49	1.68	4.60	3.90			0.64	0.71					13.8
93	M	77	71	1.65	5.00	5.10		19.00	0.36	0.72		0.39		0.47	8.6
94	M	77	63	1.70	3.90	4.10			0.64	0.51					9.5
95	M	77	65	1.70	4.70	5.30			0.42	0.57					9.4
96	M	79	72	1.66	5.50	5.20			0.54	0.50					9.9
97	M	80	69	1.67	4.60	4.50			0.58	0.49					8.8
98	M	82	80	1.87	4.60	4.50			0.60	0.58					10.9
99	M	82	73	1.74	4.20	4.20			0.66	0.59					10.4
100	M	83	50	1.56	4.60	4.50	37.10	38.40	1.25	1.14	0.69	0.69	0.72	0.79	8.1

N: number of subjects; S: sex; A: age; W: weight; H: height; I.C.d: Internal Carotid lumen at 2.5 cm after its origin; KK or: distance of the kinking from the origin of Internal Carotid Artery; S.V.: maximal systolic velocity at level of the kinking; V.B.: maximal systolic velocity before the kinking; V.A.: maximal systolic velocity after the kinking; r= right; l= left; LN=lenght neck.

TABLE II.—*Kinkings presence and percentage in two sexes (200 internal carotid arteries).*

Presence	Male	Female	KK	%
Bilateral	6	9	30	39
Left	4	8	12	32
Right	4	7	11	29
Patients	14	24		
Total KK	20	33	53	26.50

TABLE III.—*Relationship between maximal systolic velocity (V.S.) and mean velocity (divided in 3 ranges) and lumen of internal carotid arteries (with kinkings) at 1.5 cm after its origin. p>0.05.*

V.S.	Mean velocity	Mean calibre
<1 m/sec	0.81±0.10	4.58±0.45
1-1.5 m/sec	1.19±0.10	4.52±0.40
1.5-2 m/sec	1.76±0.15	4.68±0.54

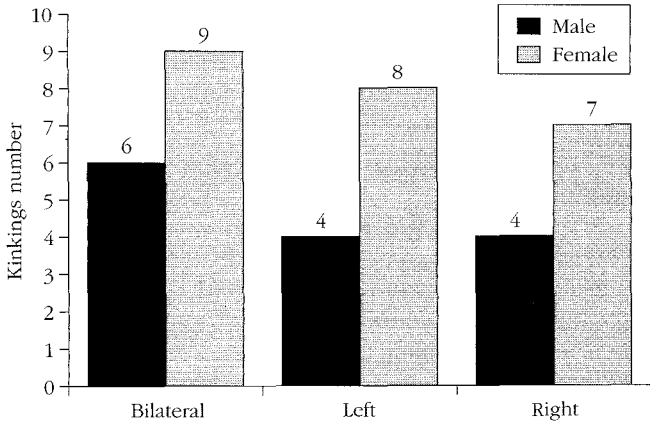

Fig. 2.—Kinkings presence in total of cases.

TABLE IV.—*Relationship between internal carotid lumen (IC cal) at 1.5 cm after its origin and kinkings (KK) presence; lumen were divided in 3 ranges.*

Side	IC cal	ICA.	KK	%
Right	3.20-4.20	22	2	8
	4.21-5.20	63	21	81
	5.21-6.20	15	3	11
Left	3.20-4.20	21	6	22
	4.21-5.20	64	20	74
	5.21-6.20	15	1	15

ICA: internal carotid arteries. p>0.05.

In 23 arteries (43% of the arteries in which a kinking was present), the maximal systolic velocity was higher than 1 m/sec (value out the normal range) at the level of the kinking. Nevertheless, no haemodynamic consequence was demonstrated distally to the kinking; the MSV distal to the kinking or in the ophtalmic branch was never reduced below 0.30 m/sec or demodulated (i.e. characterized by slow post-systolic velocimetric decrease). As concerns the statistical relationship between the frequency of kinking and the sex, 33 kinkings (right sided, left sided or bilateral) were found in 24 women, while 20 were found in 14 men. Such differences proved statistically significant (Table II, Fig. 2), as evaluated by the "t"-test method (Pt<0.05). On the contrary, there was no statistically significant correlation between kinking frequency and age, as evaluated by the coefficient r method (Pr>0.05). Eventually, the coefficient r method failed to demonstrate any statistically significant correlation between the presence of the kinkings and the weight, the height, the neck length and the body index (Pr>0.05).

Moreover, possible correlations between the lumens of the internal carotid artery (measured 1-1/2 cm from its origin) and the presence of kinkings were evaluated. The vessel lumens were sub-divided into three groups, separately in the right and in the left side, and the percentage of cases affected by carotideal kinking was calculated in each group. Nevertheless, there was no statistically significant correlation between the arterial lumen and the presence of kinking as evaluated by the coefficient r method (Pr>0.05), (Table IV).

Furthermore, the coefficient r method failed to evidence any statistically significant correlation (Pr>0.05) between the MSV at the level of the ICA origin and the presence of kinking.

Then, the systolic velocities of subjects with kinkings, subdivided into three groups (<1; 1 to 1.5; >1.5 m/sec), were correlated to vessel lumens (measured 1-0.5 cm from the vessel origin), but no statistical significance was demonstrated (Pr>0.05).

Eventually, possible correlation between the severity of kinking (we considered as severe those cases in which MSV measured at the level of the anomaly was greater than 1 m/sec) and the vessel lumen (measured as above described) was evaluat-

ed by means of coefficient r method, but no statistically significant relationship was present (Pr>0.05).

Discussion and conclusions

An analysis of the results demonstrates at first a surprisingly high frequency of carotid kinkings, i.e. 26.5% of all the arteries examined (100 right and 100 left), and 38% of all the patients considered. Such an observation seems to disagree with other studies, reporting a maximum incidence of 5%.[6] In other research, tortuosity of ICA was found in 10 cases of 36 dissected adults,[19] while Carney [20] found similar alterations in 4 fetuses out of 20. Moreover it must be stressed out that Vollmar *et al.*[7] observed convoluted enlargements of ICA in 45.6% of a healthy population.

In our opinion such a discordance can be explained by a separate analysis of the results obtained by means of different techniques. For instance, dissectory studies are not so valuable from a statistical point of view, as concerns vascular anomalies. In fact, subjects that undergo dissection constitute a dyshomogeneous population as regards age and pathological condition,[12] while post-mortem alterations, vascular collapse, and, most of all, mechanical manipulation of the cadaveric organs, could induce problems in identifying arterial anomalies.

As far as concerns arteriographic research, this could fail to evidence kinks, because of the fluttering of the image which could mask the vascular deviation.

Eventually, studies performed in the course of surgical operations are not statistically significant, considering only a selected population, affected by district vascular pathologies.

The above mentioned considerations enhance the role of ultrasonography as the primary technique in vascular anatomy.[16 17 21] This is true even if one considers most recent methods of diagnostic imaging, such as CT or angio-MR. Apart from technical considerations, the costs, the difficulty and the time required by those methods do not allow us to examine a wide normal population which is necessary for a statistical study.

Bibliographic data are rather dyshomogeneous, also because a generally accepted definition for kinkings is still lacking. Metz *et al.*[8] defined it as a tortuosity forming an angle lower than 90 between two segments of the same vessel. Nevertheless, this is not sufficient to define kinking, as not sufficient are the echographic or arteriographic methods, both because of the depth of the artery at the level of the kinkings (in our study they were located 26 mm on average from the arterial origin), and because of the superimposition of images caused by angulation of the two segments.

In our opinion it is rather difficult to define and classify the kinkings. On the basis of the velocimetric data measured in apparently normal IC arteries (mean MSV 0.51±0.14 m/sec) and in IC arteries affected by kinkings in which the MSV was lower than 1 m/sec (mean MSV 0.81±0.10 m/sec), we propose to consider the maximum velocimetric increase in the affected segment (with respect to the MSV of the ICA measured at 1.5 cm from its origin) as the most significant parameter. We, therefore, propose to consider as kinkings those tortuosities with a velocimetric increase higher than 60%. This percentage value results from the difference between the MSV of the kinking-free ICA and the MSV of the arteries affected by low severe kinkings (MSV <1 m/sec).

As for the distribution of this anomaly, it is interesting to notice that, with respect to the sex, the kinkings are significantly more frequent in women than men; this is an original finding, never reported before by other researchers.

The lack of correlation with age seems to support Carney's hypothesis [20] that such anatomic variants are due to a congenital defect, as stated also by Zanetti.[5] Even if Adachi [12] considered carotideal tortuosity as typical of aged people, it must be stressed that, in the human embryo, the ICA, after its origin from the third aortic arch and the root of the thoracic aorta, follows a tortuous course that becomes rectilinear as soon as the heart and the large vessels begin their descent into the mediastinum. Thus, it is reasonable that any factor blocking the descent of the heart might induce an incomplete enlargement of the carotid arteries, which could persist into adulthood in the form of an enlarged, tortuous artery, even bilaterally (Fig. 3 A-B).

In the light of this pathogenic hypothesis, the lack of correlation between the presence of kinkings and the weight, the height, the neck length,

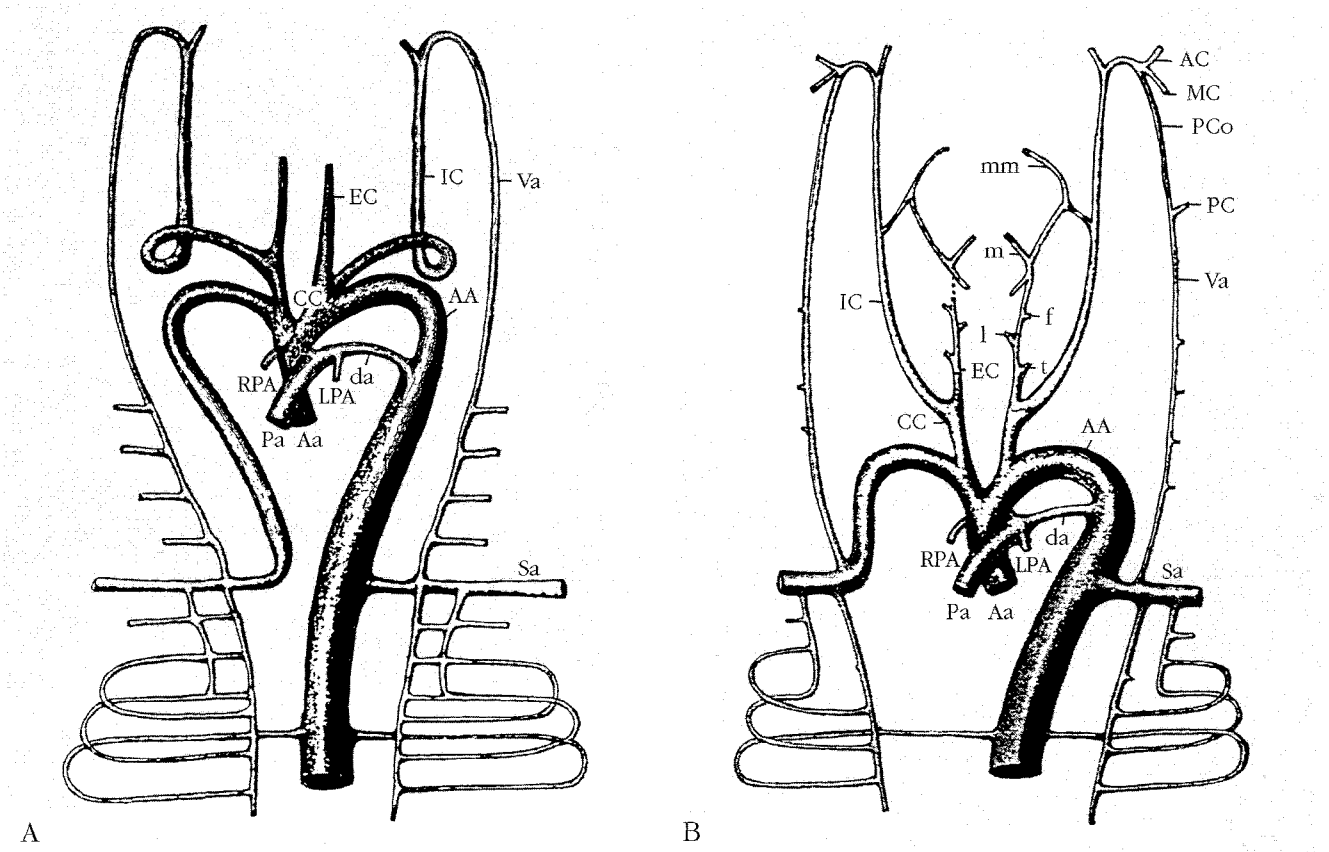

Fig. 3.—Normal developmental stages (A and B) of the aortic arch and the supra-aortic trunks of the human embryo between the fifth and the seventh week of development. In A : Pa=Pulmonary a.; Aa=aortic a; Sa=subclavian a.; RPA=right pulmonary a.; LPA=left pulmonary a.; da=ductus arteriosus; AA=aortic arch; CC=common carotid a.; EC=external carotid a.; IC=internal carotid a.; Va=vertebral a..In B : Pa, Aa, Sa, RPA, LPA, da, AA, CC, EC, IC, Va=see Fig. 3A; t=superior thyroid a.; l=lingual a., f=facial a.; m=maxillar a.; mm=middle meningeal a.; AC=anterior cerebral a.; MC=middle cerebral a.; PCo=posterior communicating a.; PC=posterior cerebral artery.

and the arterial lumen in our subjects is well-explained.

The lack of significant correlation between the presence of a kinking and the diameter of the ICA (measured 1-0.5 cm from its origin) seems to rule out a role of the arterial lumen as a risk-factor. Nevertheless, it must be remembered that some correlation was demonstrated between the arterial lumen and the atherosclerotic disease and between the latter and the presence of kinkings.[21]

On the other hand some authors considered the kinkings to be due to fibromuscular dysplasia (FMD), a degenerative process of the tunica media in which the elastic and muscular tissues are replaced by fibrous tissue.[22 23] However, FMD should lead to segmental or focal narrowings of the arterial lumen with alternating stenotic and ectasic segments;[24 25] moreover, FMD is relatively rare in comparison with the high frequency of kinkings.

Albanese *et al.*[22] reported a reduction of the elastic component in the tunica media of these vessels, particularly in the coiled portion, so considering the kinkings a malformative pathology.

The problem of aethiopathogenesis is important. Kinking, infact, is certainly a morphological abnormality, but given its frequency it could be considered as not completely pathological. Nonetheless, in 43% of the arteries in which a kinking was present (23 cases out of 53) the measured MSV exceeded 1 m/sec.

The normal IC arteries observed in the present

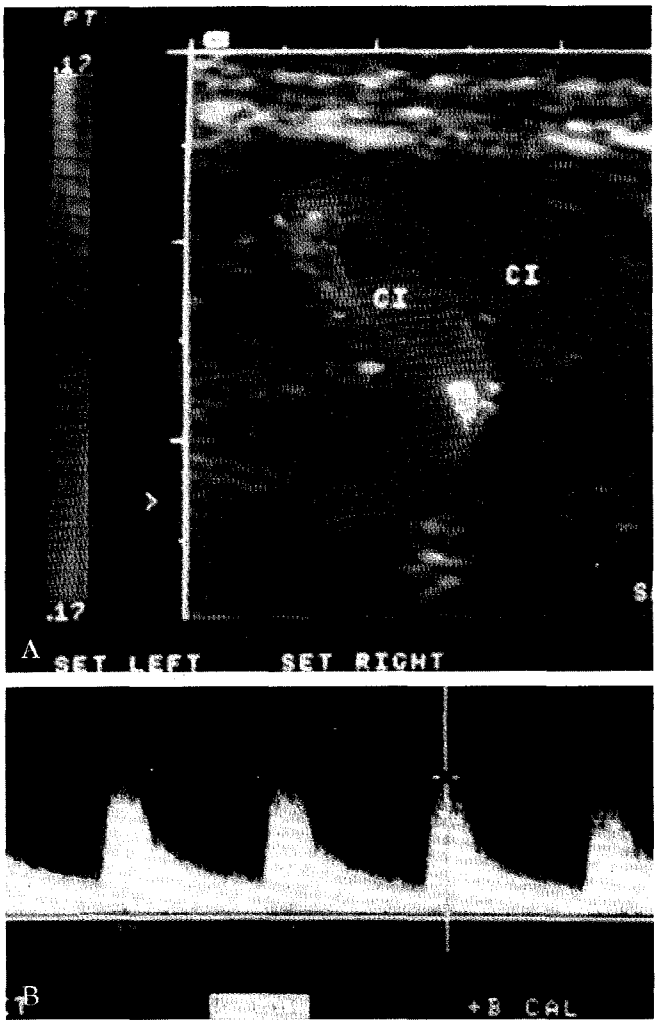

Fig. 4.—Echo color Doppler illustrating a kinking of internal carotid a (A) and systolic velocity measurement at its level (B).

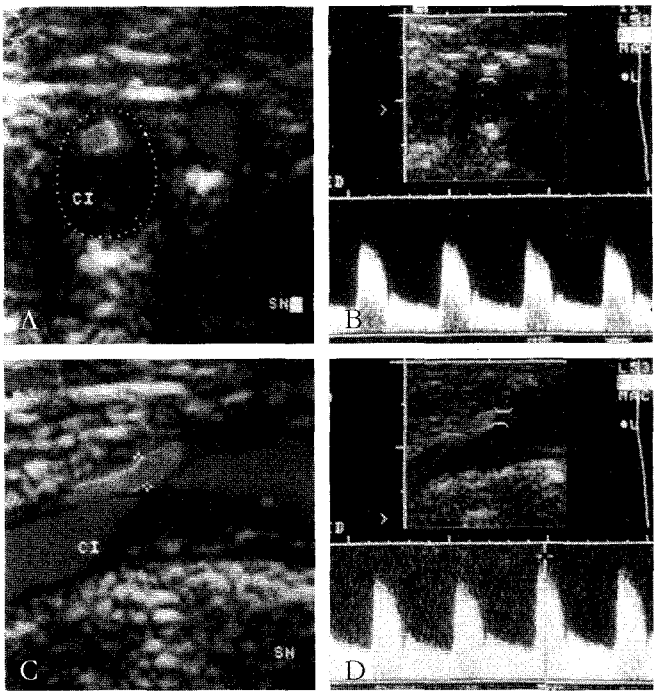

Fig. 5.—Echo color Doppler illustrating an atherosclerotic stenosis of the internal carotid a. and systolic velocity measurement at its level (case of an a 93% carotid stenosis with a residual luminal area of 5.5 mm^2 and maximal systolic velocity of 2.21 m/sec); A: transversal section; B: systolic velocity measurement; C: longitudinal section; D: systolic velocity measurement.

study showed, at 1.5 cm from the origin, a 0.51 m/sec MSV; this velocity corresponds to a 4.59 mm caliber, with a 16.46 mm^2 luminal area. If we hypothesize, in a normal artery showing these values, a stenosis reducing the lumen to 50%, at equal arterial pressure and haematocrit (haematocrit is the principal parameter able to modify the intracranial resistance [26]), the MSV would be 1.40 m/sec. A kinking determining a similar MSV value would correspond, therefore, to a 50% stenosis; nevertheless, in our patients, this value is still not able to significantly alter the blood flow. Figures 4 and 5 give an example of correlation between kinking and vascular stenosis; Figure 4 shows a kinking of the ICA with MSV=1.94 m/sec, while Figure 5 shows a stenosis of the ICA (atherosclerotic disease) with a similar MSV (2.21 m/sec), corresponding to a 83% stenosis, with a residual luminal area=5.5 mm^2. The kinkings probably induce a stenosis, but the latter generally does not induce significant haemodynamic disturbances. Nevertheless, it would be interesting to evaluate if, in some movements of the neck, the stenosis induced by the kinking would be able to modify itself, altering the blood flow.

It is not possible to rule out that pathologies related to the presence of a kinking can occur, and, in some cases, corrective surgical prophylaxis might be beneficial.

Nevertheless, in our opinion, the high frequency of kinking in asymptomatic patients does not allow to consider *tout court* this morphological aspect as a pathological condition, according to many authors,[8 23 24 27] even if some researchers report

haemodynamic disturbances induced by a remarkable tortuosity of ICA.[5 28 29] It is important to point out that an ultrasonographic study allowing a highly precise evaluation of the kinking morphology and introducing the parameter of flow velocity, is particularly suitable in identifying the boundary between normal and pathological conditions. In conclusion, the present study allows us to affirm that:

1) ICA kinking is a morphological anomaly with a high incidence (38% of normal population);

2) ICA kinking is significantly more frequent in women than in men, but it is uncorrelated with age, weight, height, neck length and body index;

3) the anatomic study has a primary role in the constitution of a normal series, necessary for control;

4) on the basis of ultrasonographic studies, it is possible to introduce a parameter (MSV), which allows a more precise definition of this anomaly.

Ultrasonography is a highly suitable technique in anatomical research on blood vessels, because it is easy, not expensive and it causes minimal disturbance for patients, thus making it possible to evaluate an extensive healthy population.

Moreover by ultrasonography it is possible to evaluate the flow velocity, which can be useful either in a clear definition of the anomaly and in distinguishing pathological from normal cases.

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