METODOLOGIA del TSA

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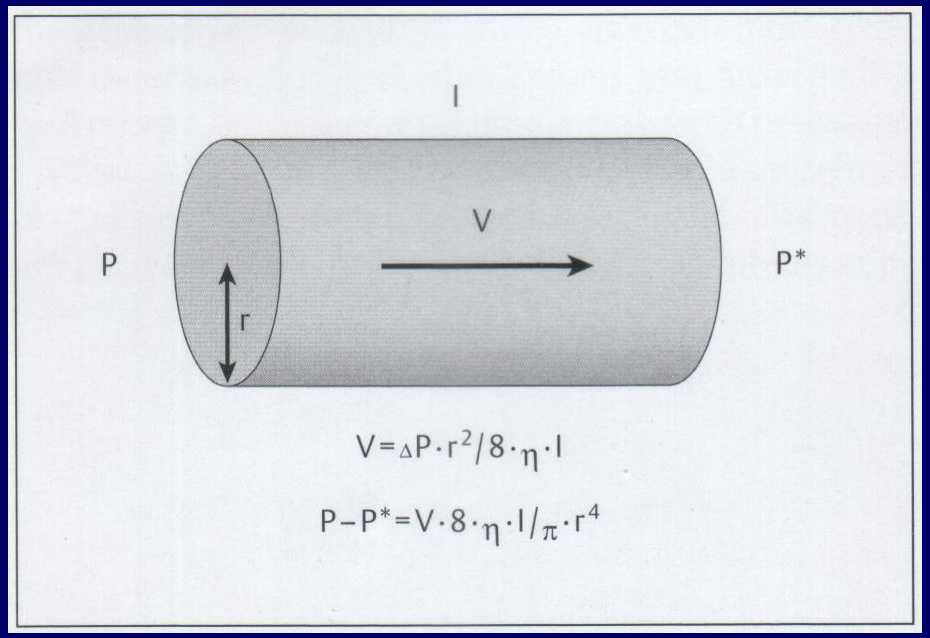
Matera

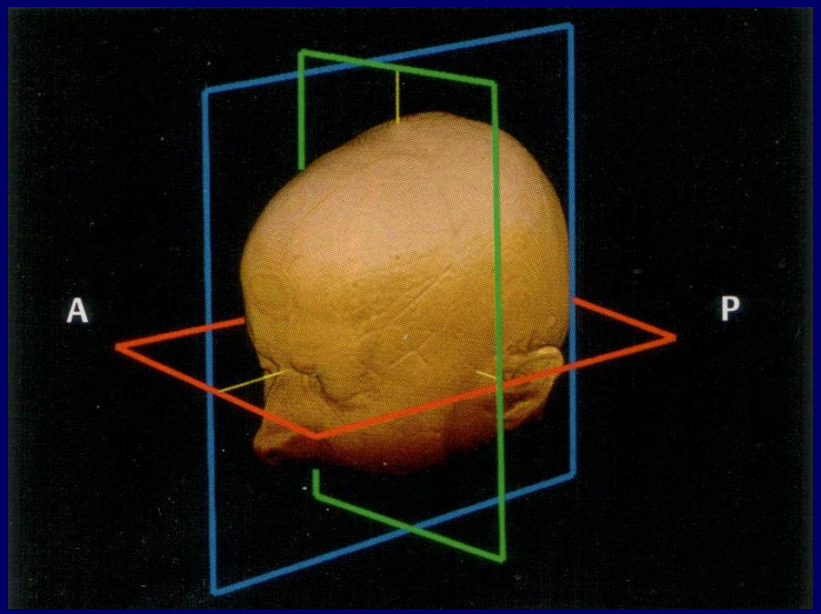
METODOLOGIA del TSA

OUTLINE

- CENNI ECOANATOMICI
- IDENTIFICAZIONE VASI CAROTIDEI E VERTEBRALI
- NORMALITA' E VARIANTI

METODOLOGIA del TSA





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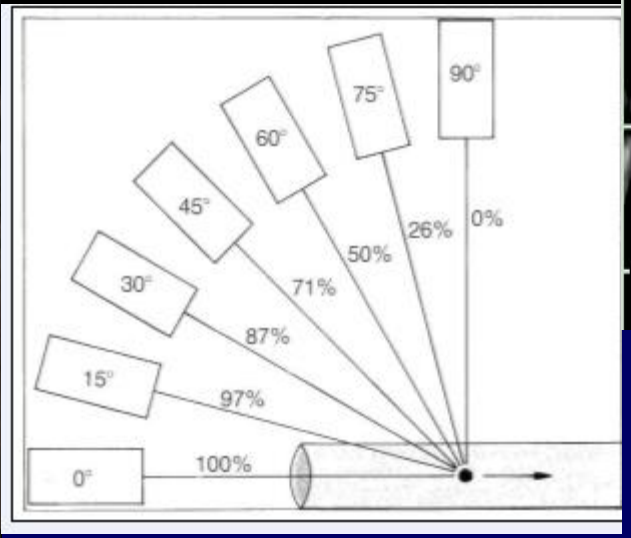

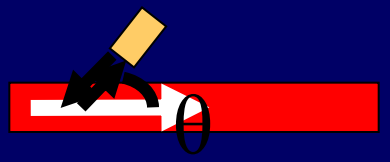

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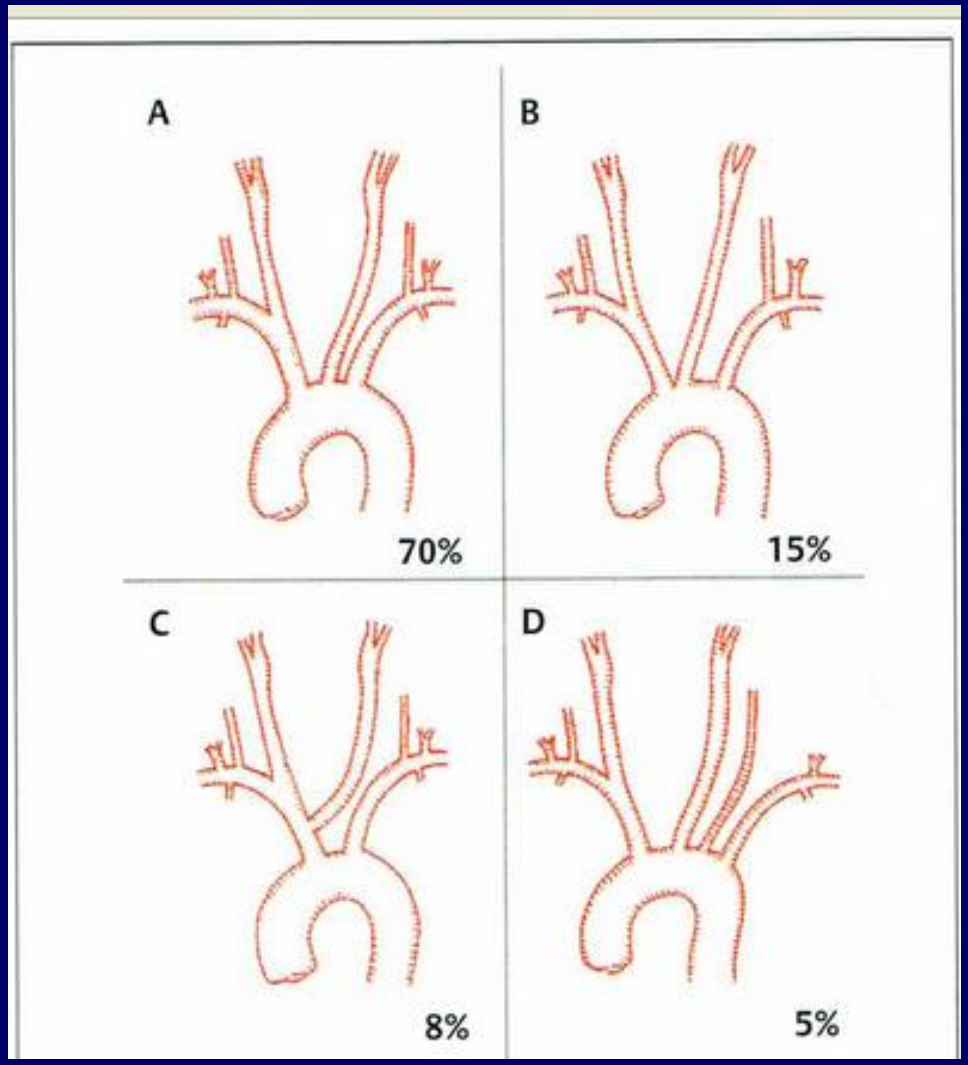
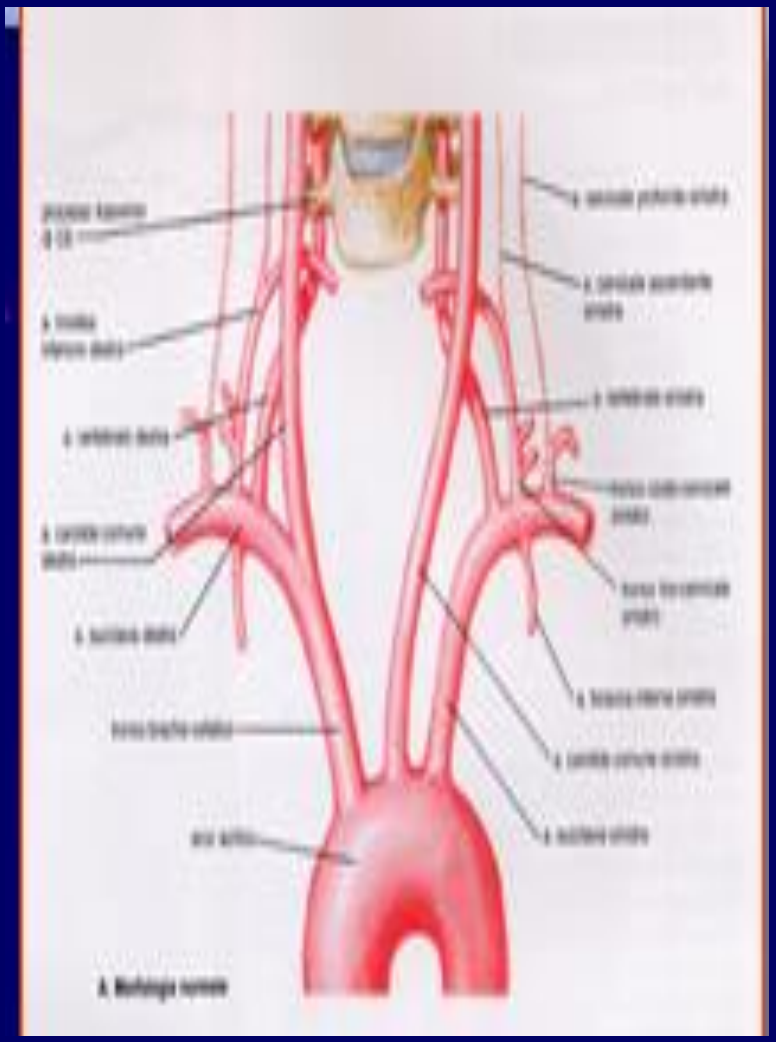
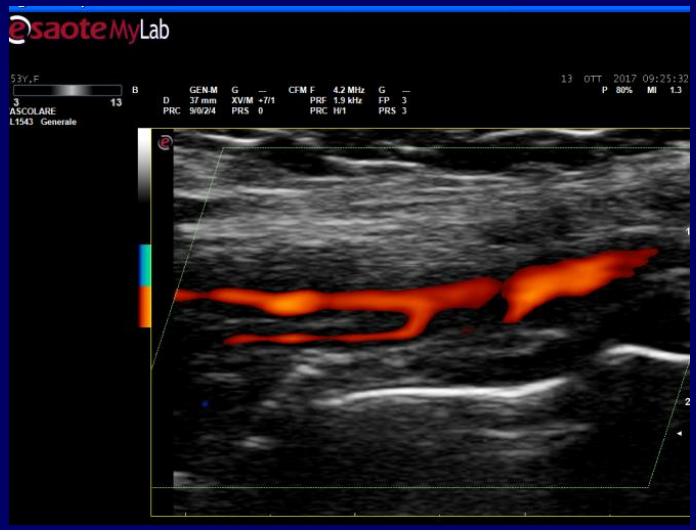




ANATOMIA DEL CIRCOLO VASCOLARE ARTERIOSO EXTRACRANICO

- **CIRCOLO ANTERIORE (CAROTIDI)**



ANATOMIA DEL CIRCOLO VASCOLARE ARTERIOSO EXTRACRANICO

CIRCOLO POSTERIORE (VERTEBRALI)

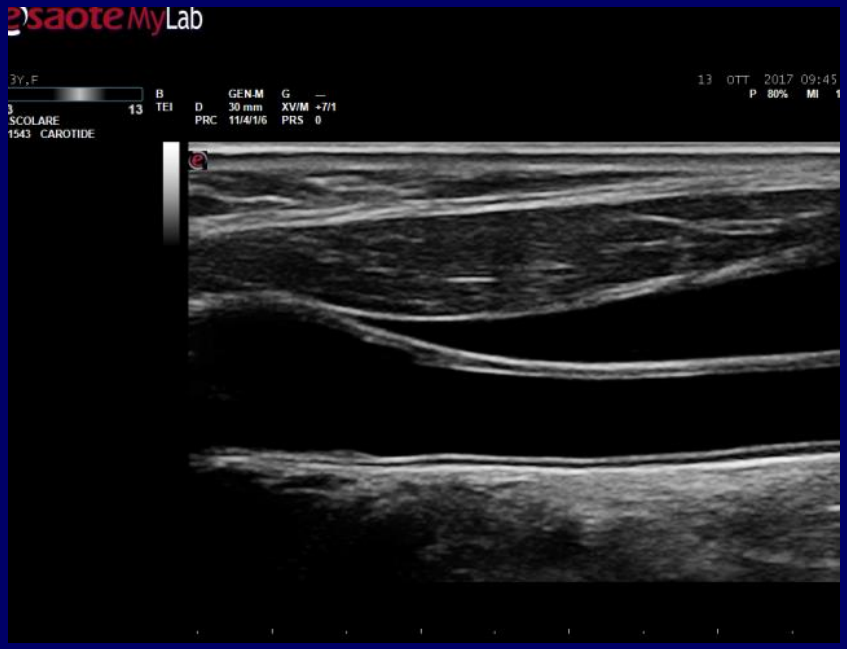
PRINCIPI GENERALI DEL TSA

- PAZIENTE SUPINO
- L'ESAMINATORE DIETRO LA TESTA DEL PAZIENTE
- ESAMINATORE COMODO CON BRACCIO POGGIATO
- TESTA PAZIENTE POGGIATA SUL LETTINO
- TRASDUTTORE LINEARE 5-10 mhZ
- PIANI LONGITUDINALI E TRASVERSALI

PRINCIPI GENERALI DEL TSA

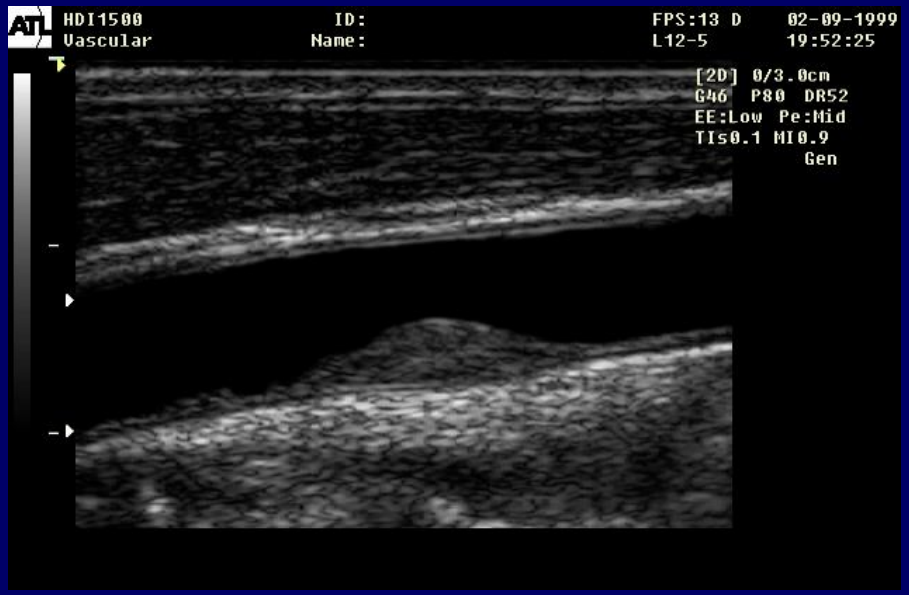
- B-MODE: ALTERAZIONI DELLA PARETE VASALE
- COLOR-MODE: RIDUZIONE DEL LUME E DIREZIONE DEL FLUSSO
- DOPPLER: VELOCITA' DI FLUSSO

ARTERIA CAROTIDE COMUNE



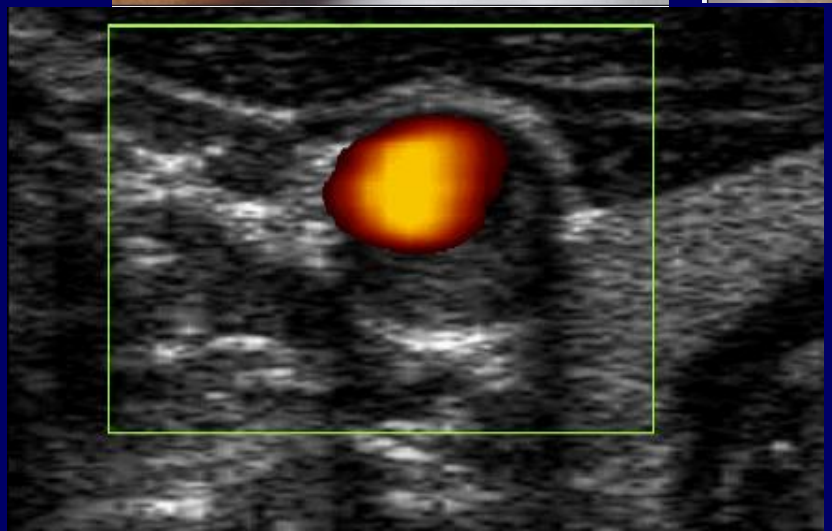
CENNI ANATOMICI

Ad eccezione delle varianti di origine non presenta frequenti altre varianti.
Elongazioni talora negli anziani. Calibro 6-7 mm



ARTERIA CAROTIDE COMUNE

IDENTIFICAZIONE DEL VASO possono essere visualizzate per oltre 5 cm prossimalmente alla biforcazione carotidea. Testa girata controlateralmente di 10-20°. Piani longitudinali e trasversali. Facile distinguerla dalla VGI



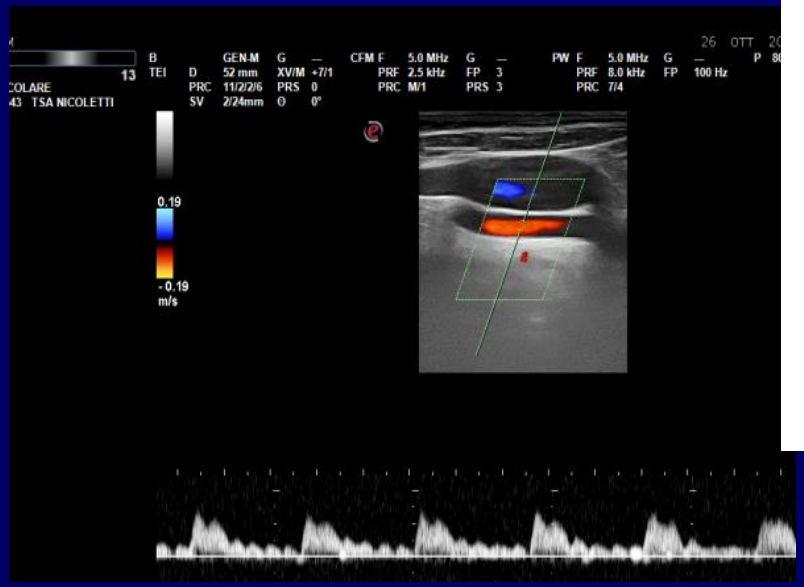
ARTERIA CAROTIDE COMUNE

Extracranial Occlusive Vascular Disease: Does Size Matter?

LOUIS R. CAPLAN, M.D. AND RICHARD BAKER, M.D.

SUMMARY A 47-year-old patient had lesser development of her left limbs and face, smaller left vascular structures, and severe occlusive disease of the left internal carotid, left subclavian, left vertebral and left iliac arteries. In 12 examples of occlusion of a carotid artery, 10/11 with unequal vascular luminal size had the occlusion on the smaller side. Of 20 patients with severe asymmetrical carotid stenosis, 13/17 with unequal size had more stenosis on the smaller side. These preliminary observations suggest that a small vessel may be more likely to occlude than its larger counterpart.

Stroke, Vol 11, No 1, 1980



Arterial occlusions: does size matter?

Louis R Caplan

Are the smaller of paired arteries more vulnerable to occlusion?

"Such knowledge as we have of these steps is derived from casual glimpses afforded when, as the outcome of one of Nature's experiments, some particular line is interfered with..."
E Garrod. *Inborn errors of metabolism*

Giannopoulos and colleagues,[1] in a paper in this issue of *J Neurol Neurosurg Psychiatry*, report three instances of lateral medullary infarcts in young patients (average age 38 years) associated with occlusion of ipsilateral hypoplastic vertebral arteries (*see page 987*).[1] All three patients had high blood pressure and one had hyperlipidaemia.

The two vertebral arteries are very often asymmetrical. Chuang *et al* investigated 191 patients with acute ischaemic stroke and found that 11.5% had a unilateral hypoplastic vertebral artery.[2] They found a significant relationship between vertebral artery hypoplasia and ipsilateral posterior circulation infarction.[2] In the New England Medical Center Posterior Circulation Registry, we also noted that in patients with infarcts involving the proximal intracranial territory fed by the vertebral arteries (medulla and posterior inferior cerebellum), occlusive lesions favoured the smaller vertebral artery ipsilateral to the infarcts.[3] These observations raise the larger question—among paired arteries, are the smaller arteries more likely to occlude? And, if so, why? Does size matter and how?

I became interested in this question more than a quarter of a century ago, stimulated by a single patient.[4] A 47-year-old woman had attacks of left monocular visual loss and a right inferior quadrantanopia. She had claudication involving her left calf. Blood pressure was 180/90 mm Hg in the right arm. Examination showed that her left hemibody and limbs were appreciable smaller than the right counterparts. She had no left hand or foot pulse and these extremities were cool. Angiography showed occlusions of her left internal carotid and left common iliac artery and severe stenosis of the left subclavian and the left vertebral artery. All of the left-sided arteries were considerably smaller than those on the right. The right-sided arteries were all normal. This clearly was an experiment of nature. All her vessels were supplied by the same bloodstream and yet all the smaller arteries of pairs were severely compromised while the larger counterparts were spared.

In order to formally study the issue of size, I investigated 20 patients in whom one internal carotid artery (ICA) was occluded or severely stenotic while the contralateral artery was spared.[4] Among 12 patients with unilateral ICA occlusions, 10/11 with unequal arterial size had the occlusion on the smaller side. Among 20 patients with severe asymmetrical ICA stenosis, 13/17 with unequal sized arteries had the stenosis on the smaller side. Most of the patients had vascular risk factors. Luminal size varied considerably from 2.8 to 9.5 mm.

These data, along with the observations of Giannopoulos *et al*[1] and Chuang *et al*,[2] lead to the inescapable conclusion that size does matter. The smaller of paired arteries are more vulnerable to occlusion, especially if vascular risk factors are present. The main question is why? Nature may be suggesting an important lesson in pathophysiology, if we could only unlock the explanation.

Clearly the explanation cannot be size alone as many intracranial arteries are smaller than the hypoplastic or smaller arteries identified in these studies, and they are not predisposed to occlude. The answer must lie somehow in the physics of blood flow and sheer forces. There must be an interaction between blood pressure, blood constituents and the rheology and physics of blood flow at various arterial locations that explains the data.

Lastly, I feel compelled to mention how often seeing single patients leads to observations and ideas that trigger investigations into basic principles. Keeping active at the bedside is a critical stimulus to new ideas. Case reports, an n of 1, are still extremely important in generating ideas and formal research. All of the answers do not lie in large analyses and meta-analyses of lumped, often heterogeneous, patients. There is great value in studying in depth the individual patient at the bedside and in the laboratory.

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doi: 10.1136/jnnp.2006.110205

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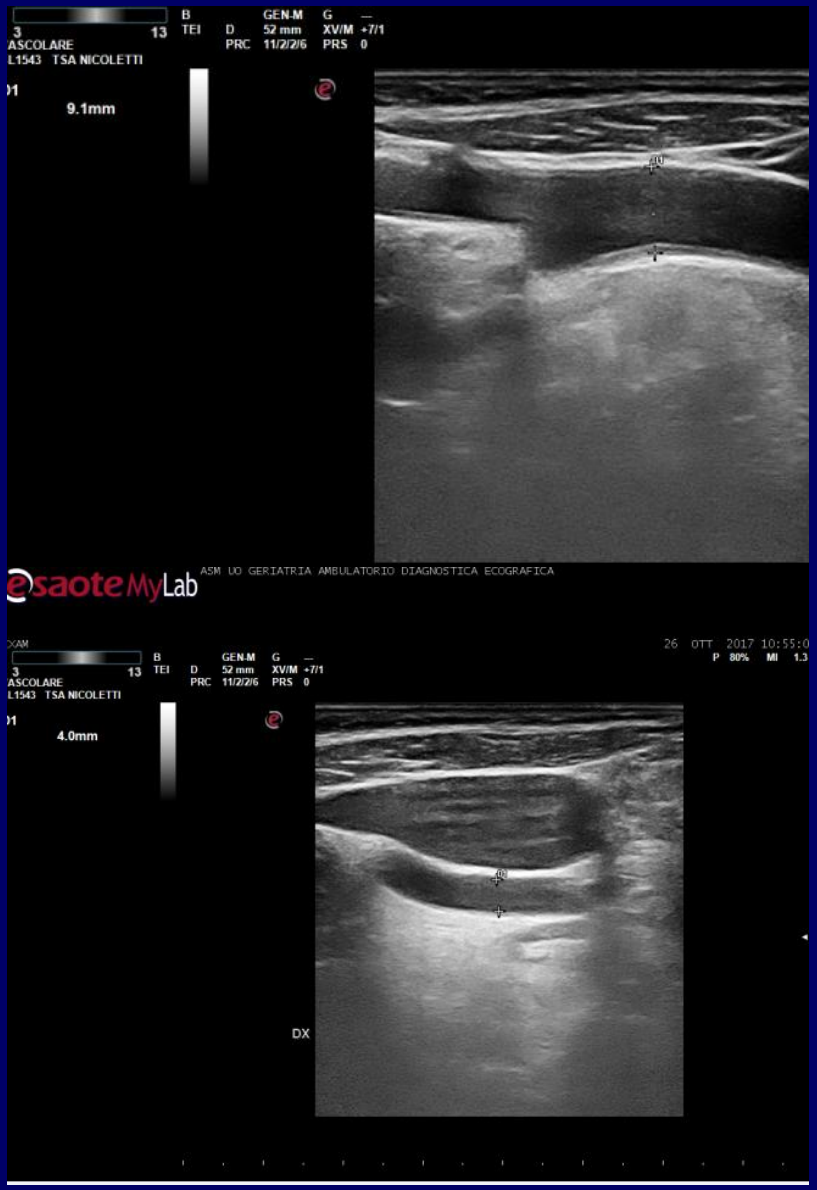
Competing interests: None.

REFERENCES

1 Giannopoulos S, Markolis S, Kosmidou M, *et al*. Lateral medullary ischaemic events in young adults with hypoplastic vertebral artery. *J Neurol Neurosurg Psychiatry* 2007;78:987–9.
2 Chuang YM, Huang YM, Hu HH, *et al*. Toward a further elucidation of the role of vertebral artery hypoplasia in acute ischaemic stroke. *Eur Neurol* 2006;55:193–7.
3 Caplan LR, Wityk RJ, Glass TA, *et al*. New England Medical Center Posterior Circulation Registry. *Ann Neurol* 2004;56:389–98.
4 Caplan LR, Baker R. Extracranial occlusive vascular disease: does size matter. *Stroke* 1980;11:63–6.

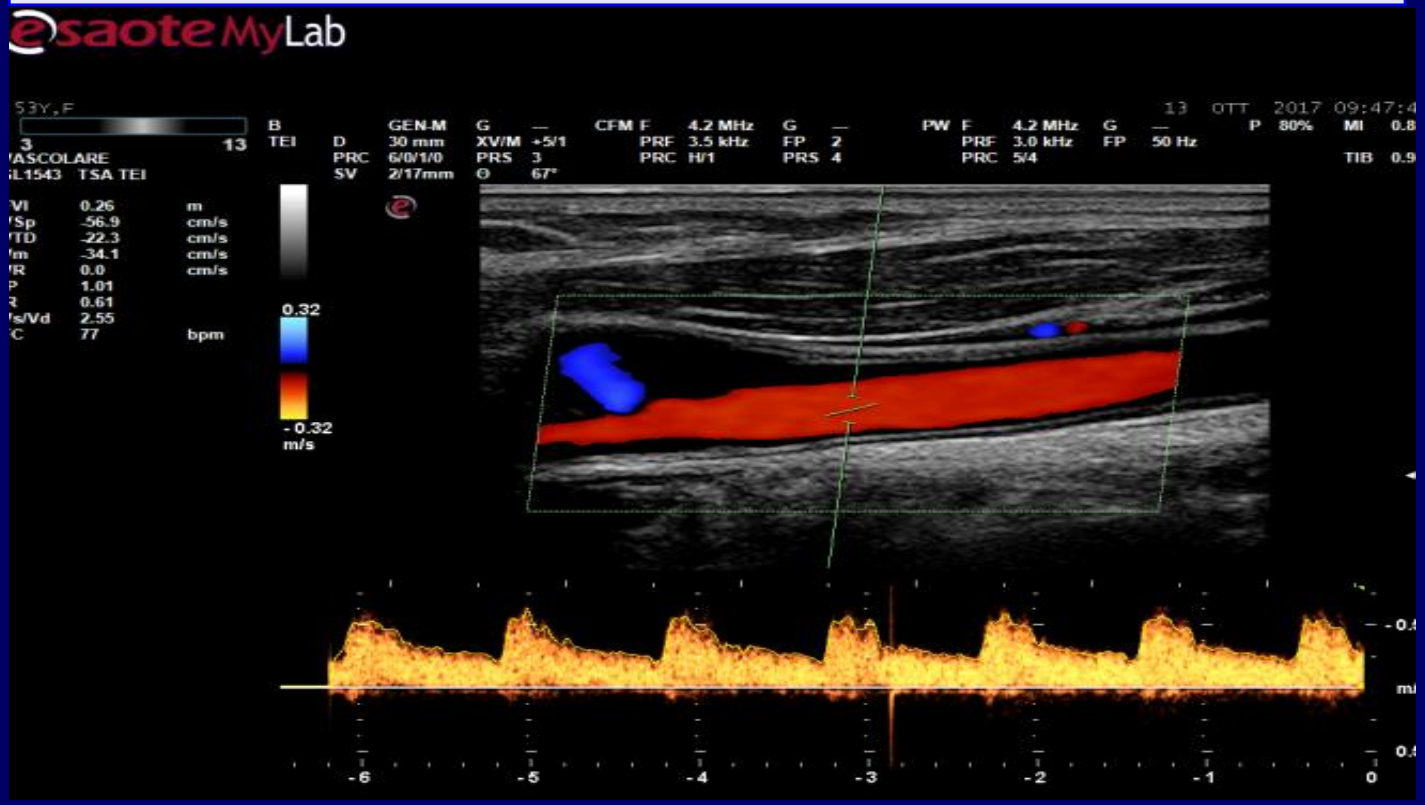
ARTERIA CAROTIDE COMUNE



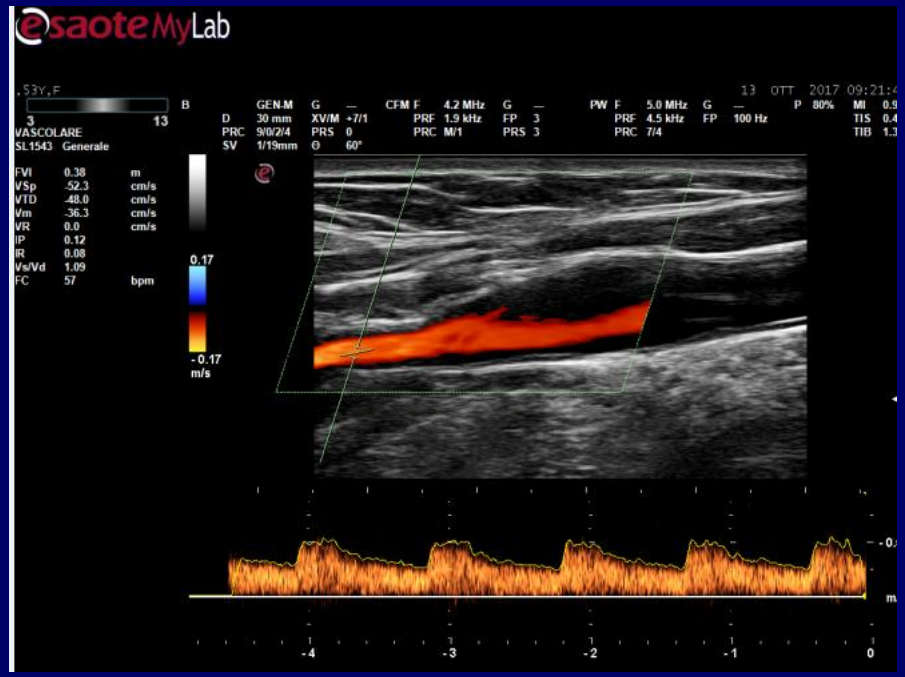
ARTERIA CAROTIDE COMUNE

VALORI NORMALI flusso laminare.

	VPS ± DS (cm/sec)	VPD± DS (cm/sec)	bibliografia
ACC	96±25	26±6	Schoning 1994

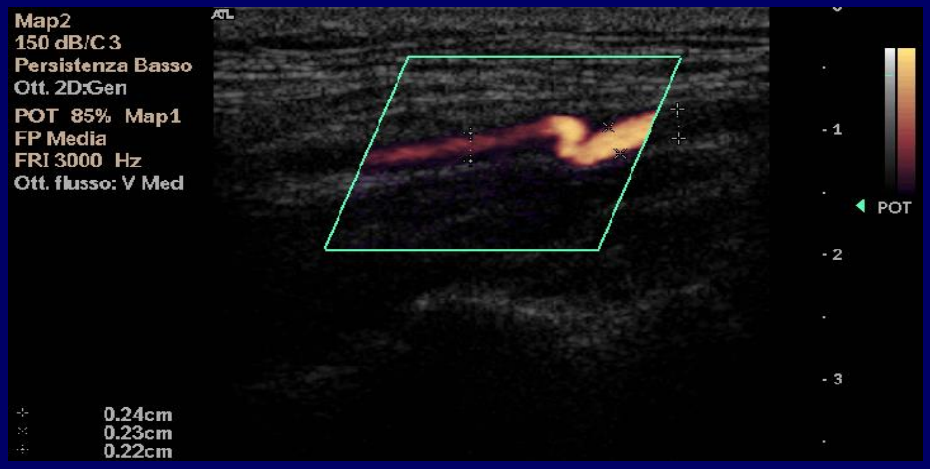


ARTERIA CAROTIDE INTERNA



CENNI ANATOMICI

A livello della cartilagine tiroidea la ACC si divide in ACI e ACE. La ACI dorso laterale (90%). Elongazioni talora negli anziani. Ipoplasie rare ma con implicazioni cliniche. Calibro 4-5 mm



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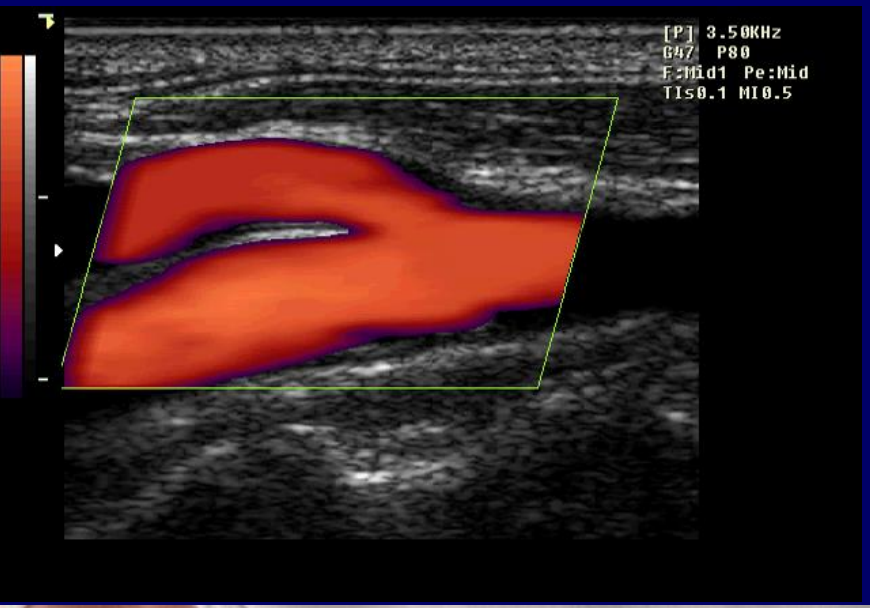
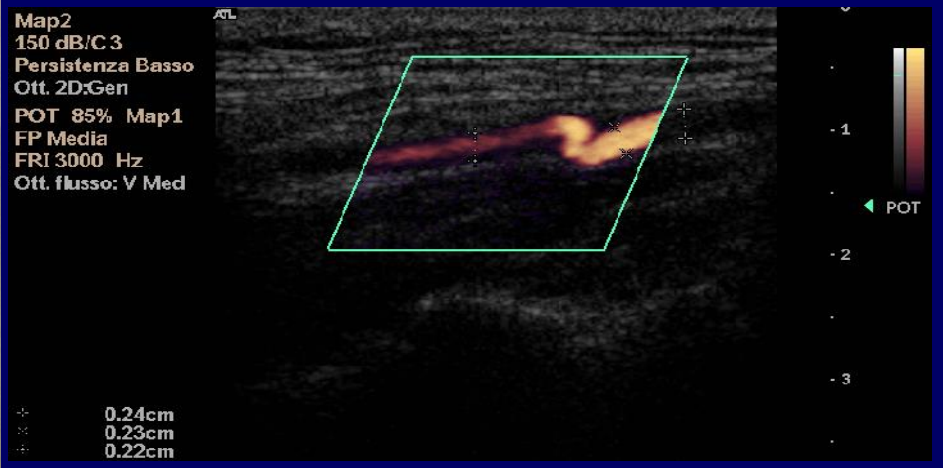


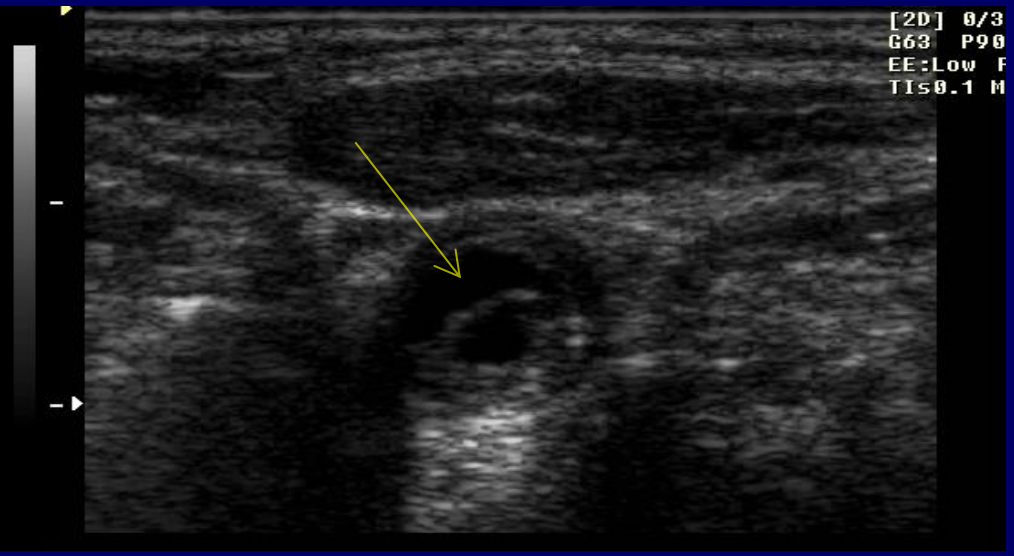
IDENTIFICAZIONE DEL VASO nel 30% è possibile visualizzarla insieme alla ACE. Proseguendo distalmente sulla comune si trova la biforcazione e quindi la ACI . Si segue distalmente per non perdersi coiling o kinking.



ARTERIA CAROTIDE INTERNA

IDENTIFICAZIONE In sospetta dissecazione l'insonazione assiale permette di seguirla ancora più distalmente.

DISSECAZIONI
In presenza di ischemia nel territorio carotideo sensibilità e specificità > 90%



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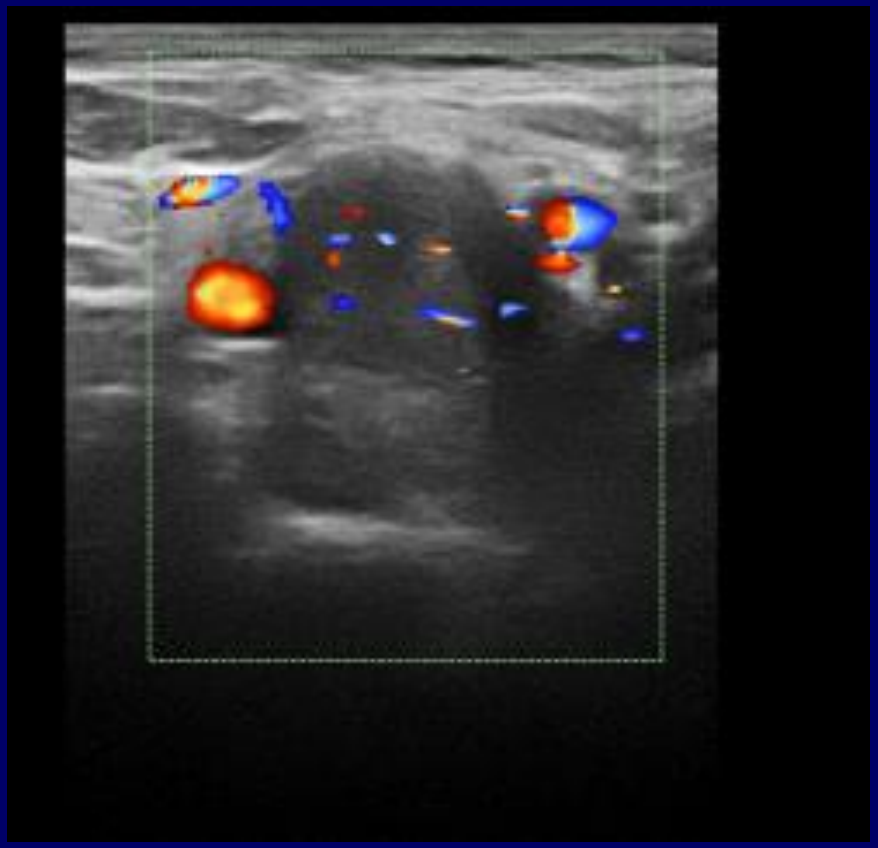


- **TUMORI DEL GLOMO CAROTIDEO**

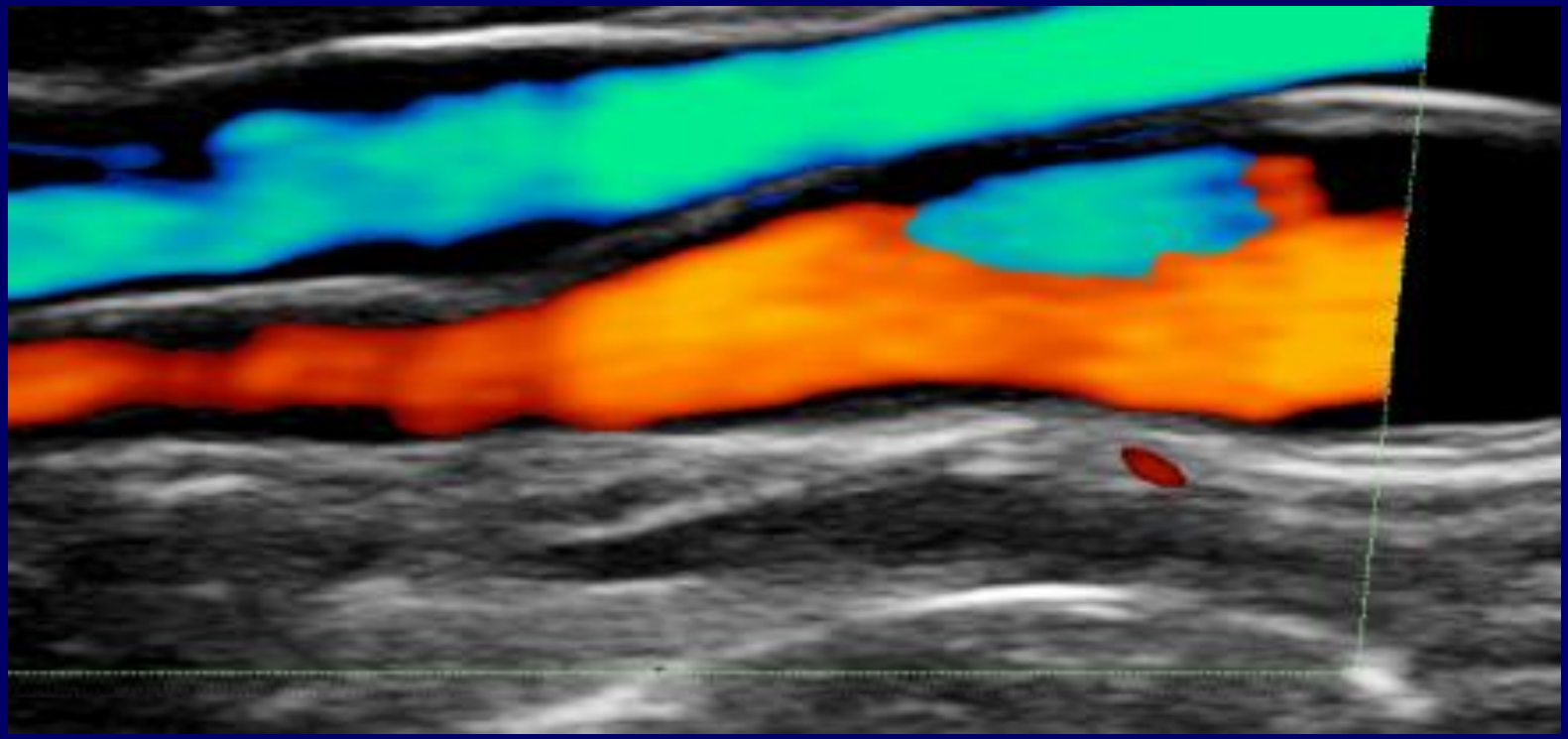
Spostano la ACE anteromedialmente, la ACI posterolateralemente e la VG posteriormente. Può causare compressione ACI e ACE ma non infiltrano.
Flussi a basse resistenze.

CAROTIDE INTERNA/ESTERNA

ACI	ACE
• dorsolaterale profonda(sonda verso la colonna)	• Superficiale (sonda verso la ACI controlaterale)
• No rami	• Si rami
• pattern doppler	• pattern doppler
• no risposta alla compressione della temporale	• risposta alla compressione della temporale

ARTERIA CAROTIDE INTERNA

VALORI NORMALI Il flusso all'origine può essere meno laminare a causa dell'allargamento del calibro e può essere visibile un apparente flusso invertito. Successivamente il flusso diventa laminare.



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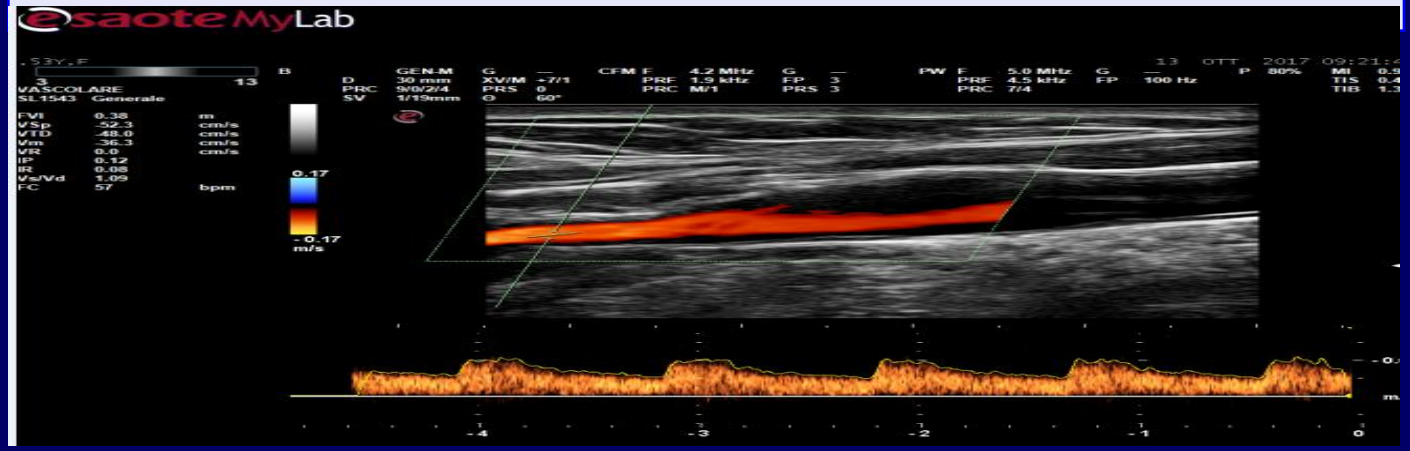
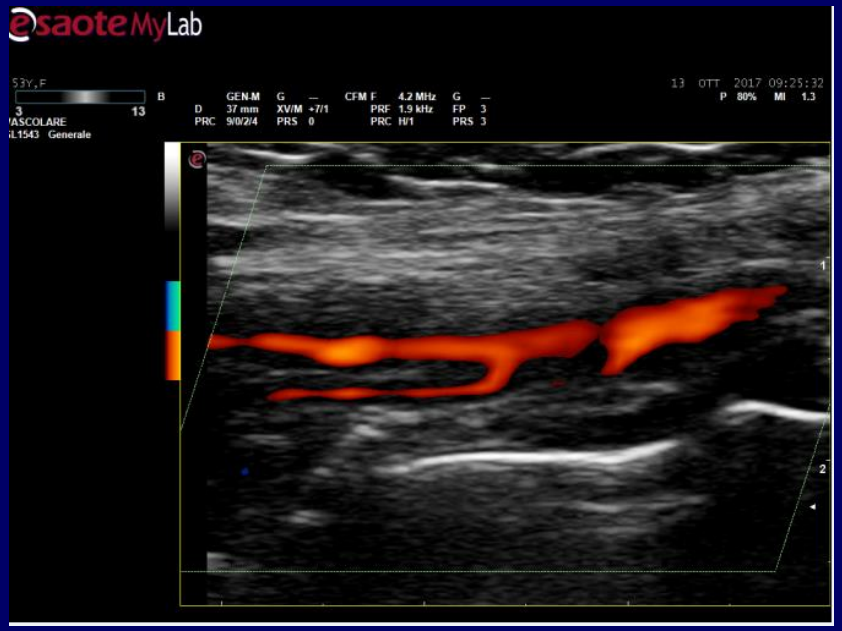
VALORI NORMALI Il flusso all'origine può essere meno laminare a causa dell'allargamento del calibro e può essere visibile un un apparente flusso invertito. Successivamente il flusso diventa laminare.

	VPS ± DS (cm/sec)	VPD± DS (cm/sec)	bibliografia
ACI	66±16	26±6	Schoning 1994

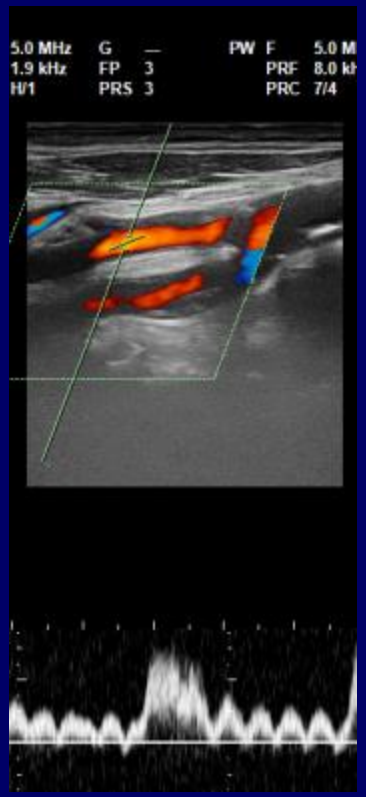


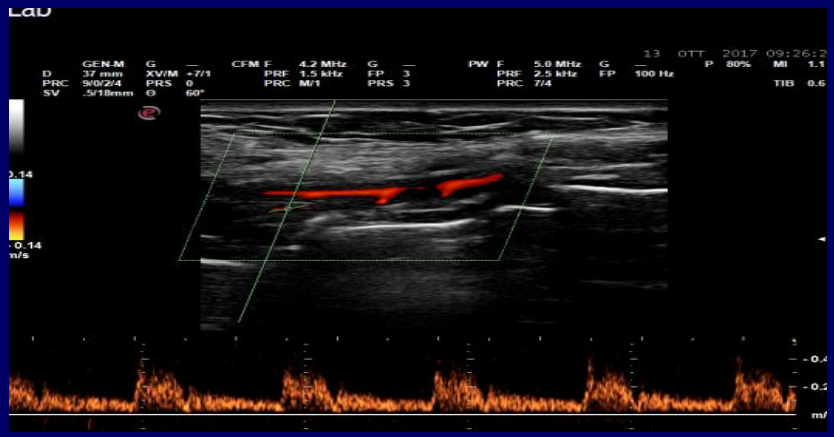
ARTERIA CAROTIDE ESTERNA



CENNI ANATOMICI

Irrora la faccia e il collo. Dà rami, il primo arteria tiroidea superiore.





IDENTIFICAZIONE DEL VASO

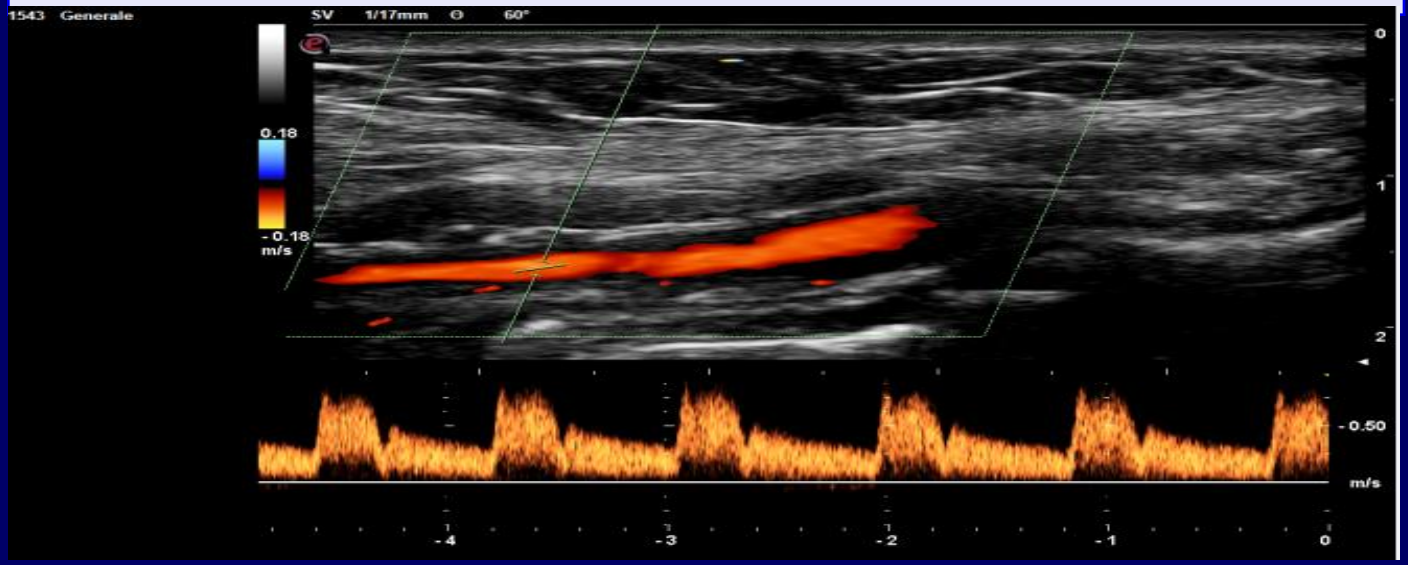
Compressione della temporale al davanti del trago.

ARTERIA CAROTIDE ESTERNA

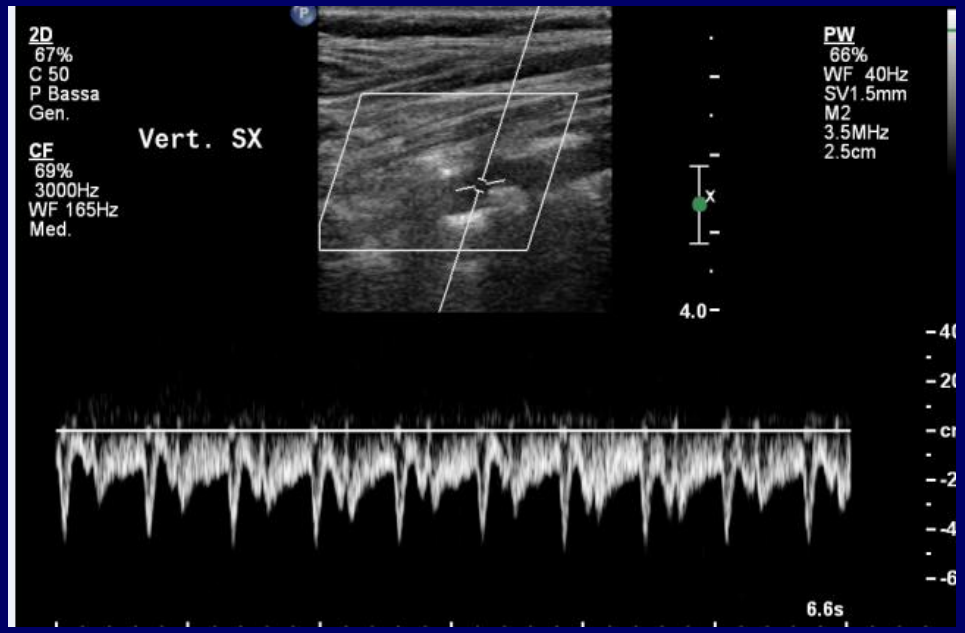
VALORI NORMALI picco sistolico più aguzzo vs interna.

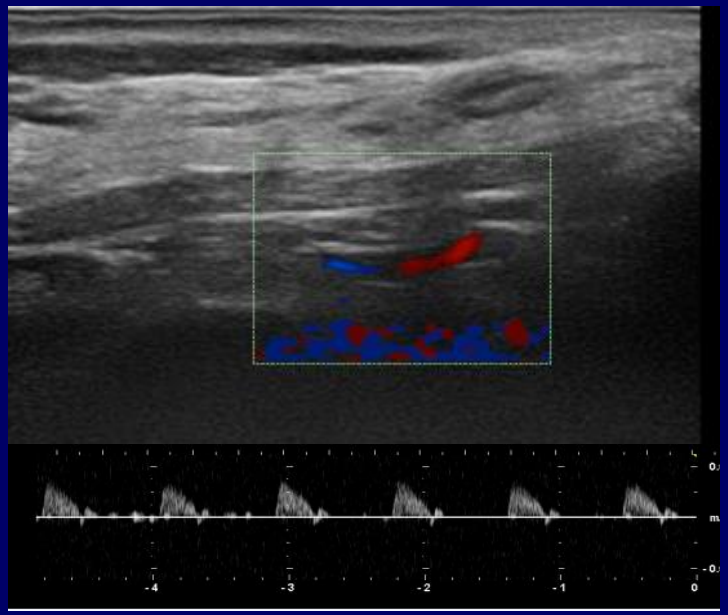
Incisura sisto diastolica.

	VPS ± DS (cm/sec)	VPD± DS (cm/sec)	bibliografia
ACE	83±17	17±5	Schoning 1994



ARTERIA OCCIPITALE



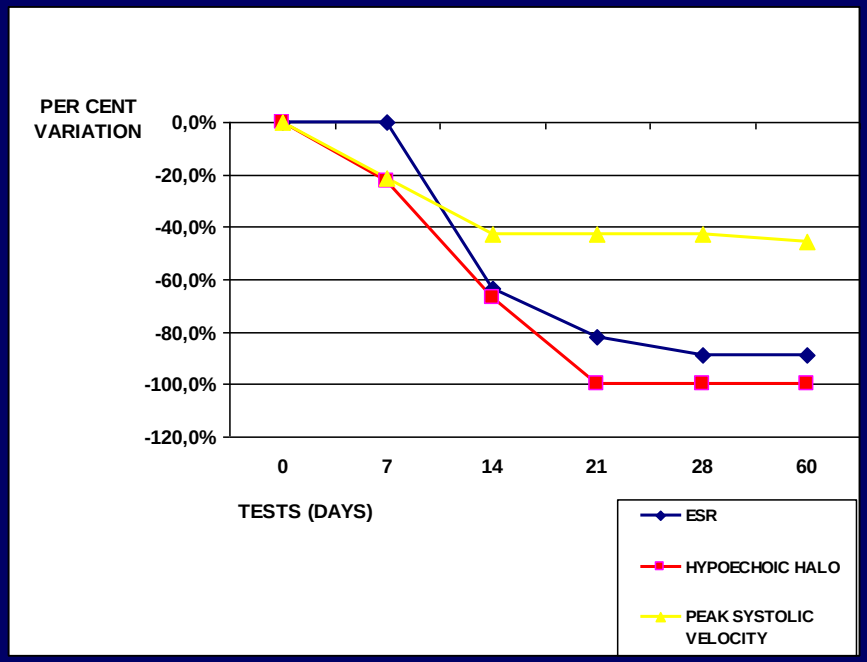


Le anastomosi tra i rami muscolari dell'A. Occipitale ed i rami muscolari dell'AV costituiscono il più importante collegamento extracranico tra il sistema carotideo e quello vertebro-basilare

ARTERIA TEMPORALE

- **Malattie infiammatorie (arterite di Takayasu, arterite gigantocellulare)**

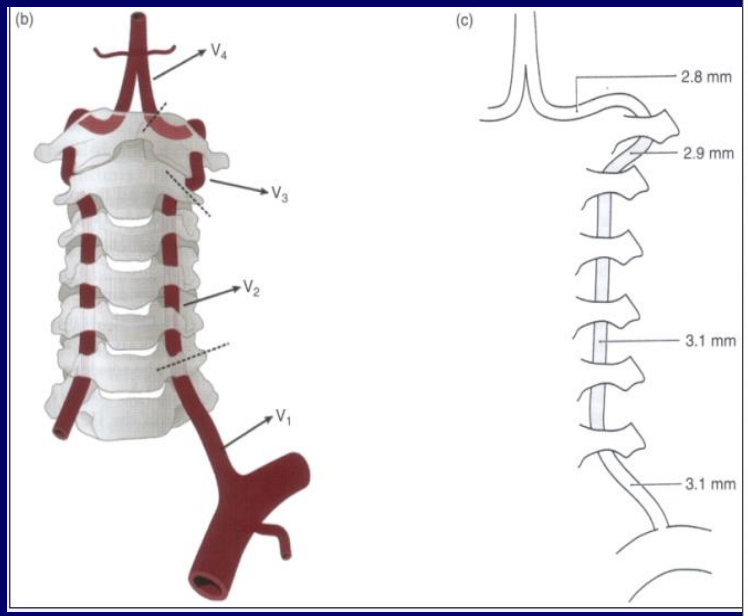
COLOUR DUPLEX ULTRASONOGRAPHY IN THE MANAGEMENT OF GIANT CELL ARTERITIS
Giuseppe Nicoletti[1], Giovanni Ciancio[2], Salvatore Tardi[1], Ignazio Olivieri[2]
Clinical heumatology2003

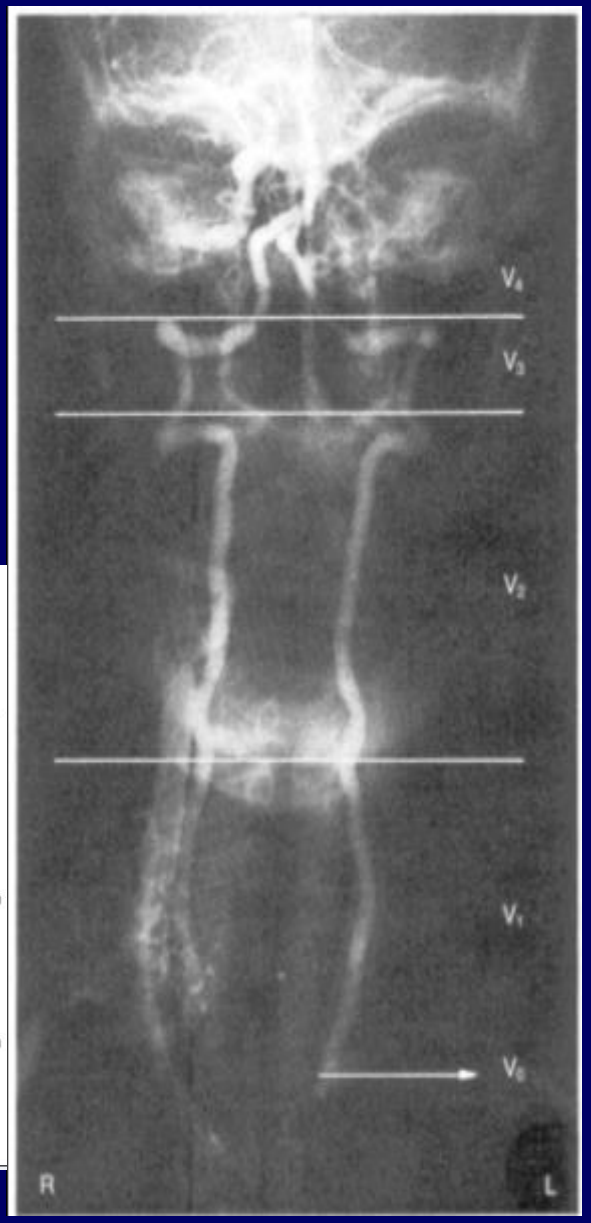


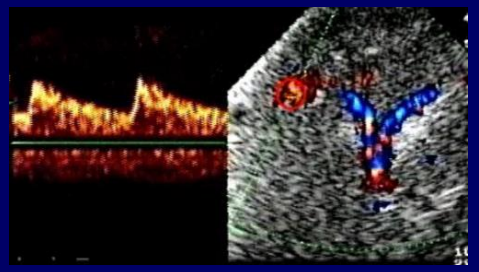
ARTERIA VERTEBRALE

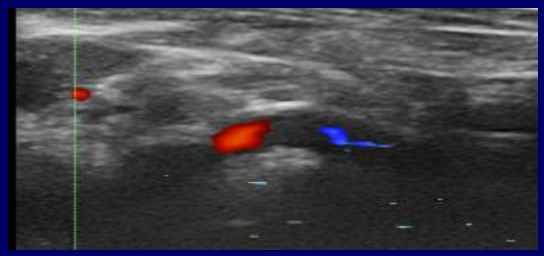
CENNI ANATOMICI

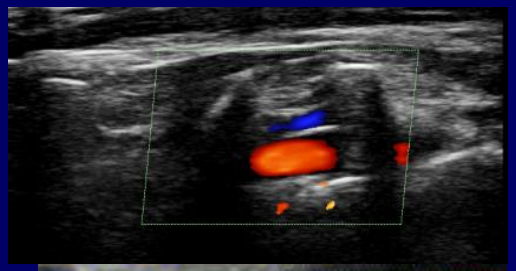
Origine dalla succlavia.
Asimmetria di calibro.
V0 origine
V1 tratto pretrasversario
V2 tratto intertrasversario
V3 tratto atlantoideo
V4 tratto intracranico

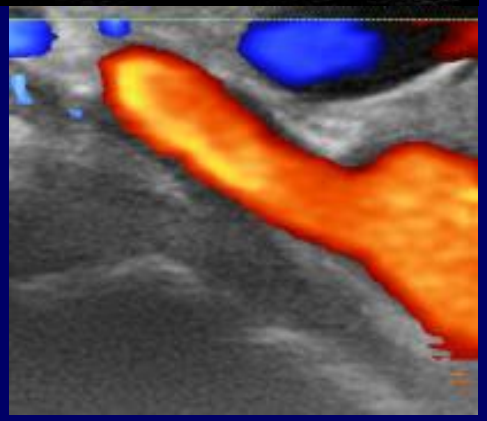












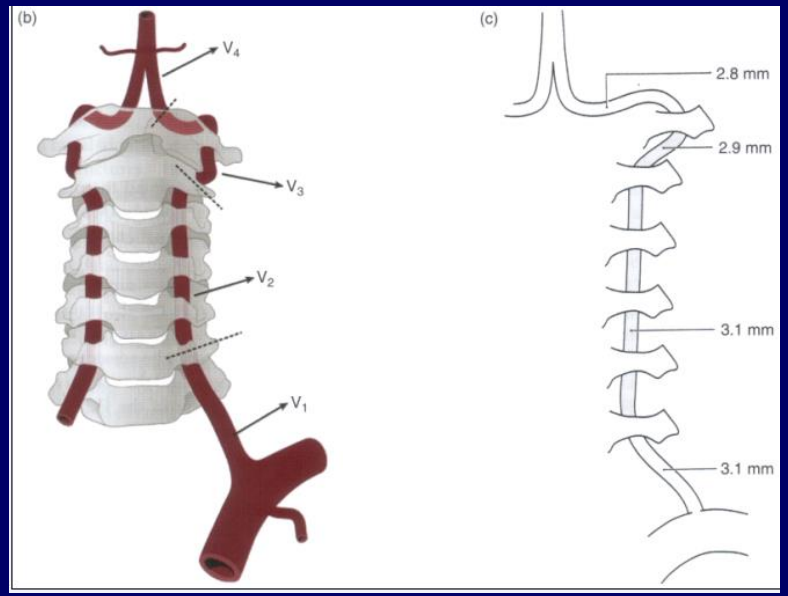
ARTERIA VERTEBRALE EXTRACRANICA

V0/V1

CENNI ANATOMICI

Origine dalla succlavia. 5% sx origina dall'arco. Asimmetria di calibro. Ipoplasia<2 mm o < 40ml/min. Insonazione della sx può presentare difficoltà per maggiore profondità origine da arco o da succlavia inferiormente.

V1 insonato facilmente



Vertebral artery hypoplasia

A predisposing factor for posterior circulation stroke?

Fabienne Perren, MD; Davide Poglia, MD; Theodor Landis, MD; and Roman Sztajzel, MD

Abstract—We determined in 725 sequentially admitted first-ever stroke patients, using color-coded duplex flow imaging of the V2 segment, whether vertebral artery hypoplasia (VAH) (diameter ≤ 2.5 mm) was more frequent (13%) in posterior circulation territory infarction (247) than in strokes in other territories (4.6%). This difference is significant ($p < 0.001$), whereas all other risk factors (hypertension, hyperlipidemia, diabetes, smoking) were equally ($p > 0.05$) distributed. Patients with VAH may be predisposed to stroke in the posterior circulation.

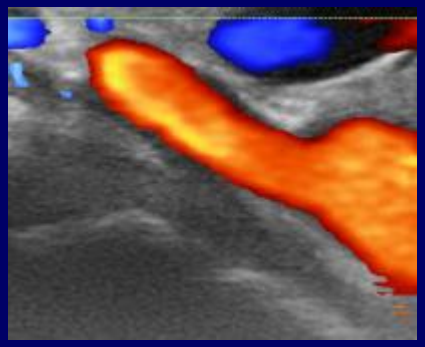
NEUROLOGY 2007;68:65–67

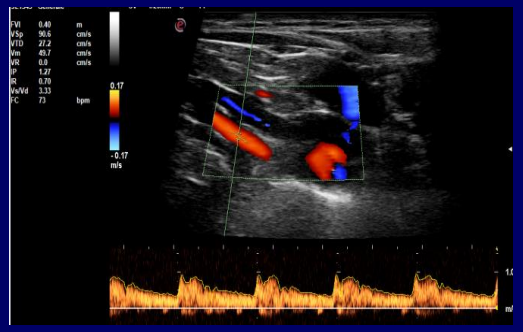
Vertebral artery hypoplasia (VAH) was described in the 19th century.[1] Congenital variations of the caliber of the vertebral arteries or asymmetry of these vessels are not infrequent.[1,2] VAH with caliber discrepancies of more than 1:1.7 were observed in up to 10% of normal individuals.[3] There is no general agreement as to the definition of VAH. Operational definitions of VAH vary between diameters of less than 2 to less than 3 mm or an asymmetry ratio of equal or greater than 1:1.7.[3-6]

Given the importance of these vascular variations, it is astonishing how little is known about their clinical relevance. In other words, is VAH a risk factor for strokes in the posterior cerebral

Methods. Seven hundred twenty-five patients (443 men, 282 women; mean age 67.11 years, ranging from 21 to 96 years) sequentially admitted for a first acute stroke confirmed by imaging (CT or MRI) during the years 2001 through 2004 had a full ultrasound examination of the extra- and intracranial arteries. Patients with previous stroke or TIAs, other cerebral lesions, occlusion, stenosis, or atheromatosis of the vertebral arteries or those in whom a comprehensive ultrasound examination of the vertebral arteries was not possible were excluded.

Duplex ultrasound examination of the vertebral arteries was performed using a 7.5-MHz linear array transducer for segments V1 and V2 and a 2-MHz device for segments V3 and V4 (Acuson; Sequoia). Diameters of the vertebral arteries were determined in the intertransverse segment V2 (situated between C2 and C6) of the extracranial vertebral artery (due to technical and anatomic limitations, precise measurement of the caliber of the vertebral artery is possible only in the V2 seg-

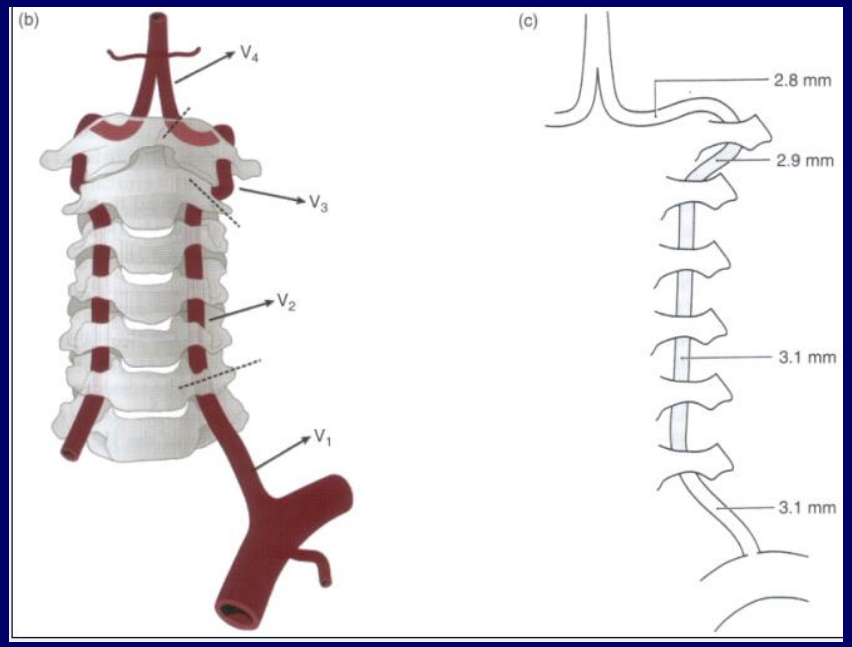


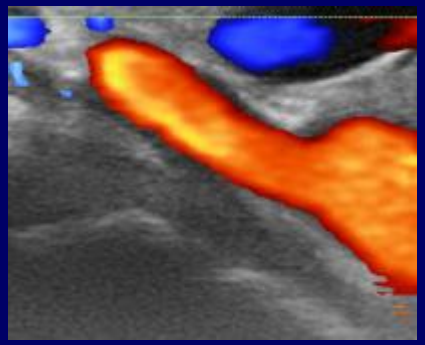


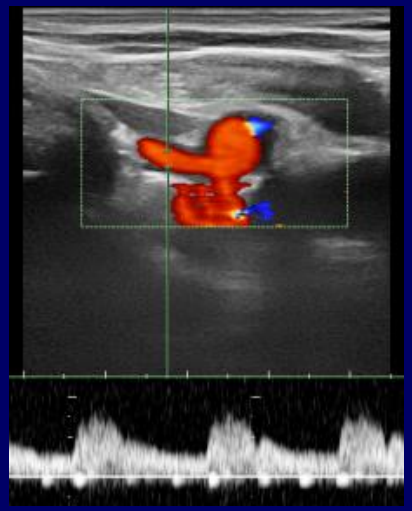
ARTERIA VERTEBRALE EXTRACRANICA

V0/V1
IDENTIFICAZIONE DEL VASO

Da V2 si scende prossimalmente.
Rispetto a V2 il V1 è più superficiale ed ha un decorso meno lineare.
Scendendo ancora si ha V0 sede di aterosclerosi.





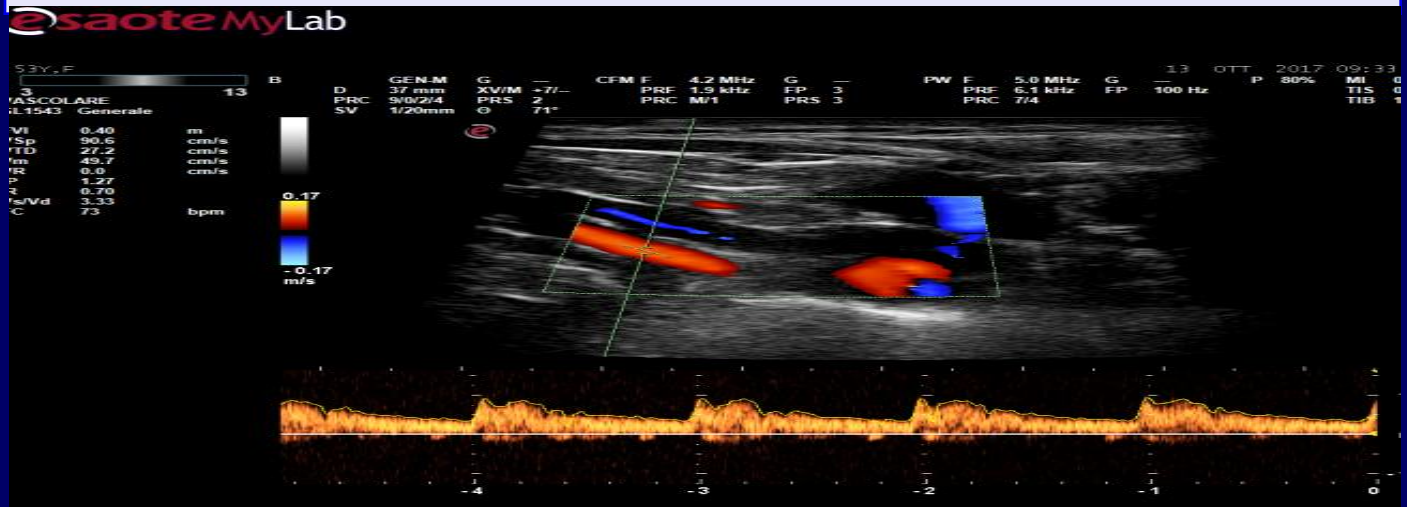


ARTERIA VERTEBRALE EXTRACRANICA

V0/V1

VALORI NORMALI all'origine lo spettro doppler può mostrare turbolenza senza significato patologico. Poi laminare.

	VPS ± DS (cm/sec)	VPD± DS (cm/sec)	bibliografia
V0 dx	66±18	16±5	Kuhl 2000
V0 sx	61±17	16±5	
V1 dx	60±14	16±5	
V1 sx	58±10	17±5	

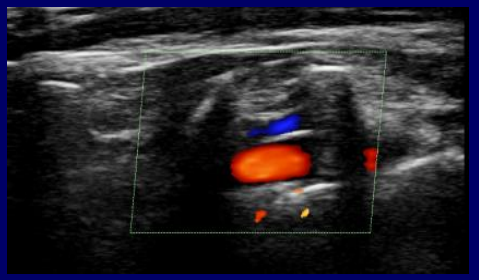


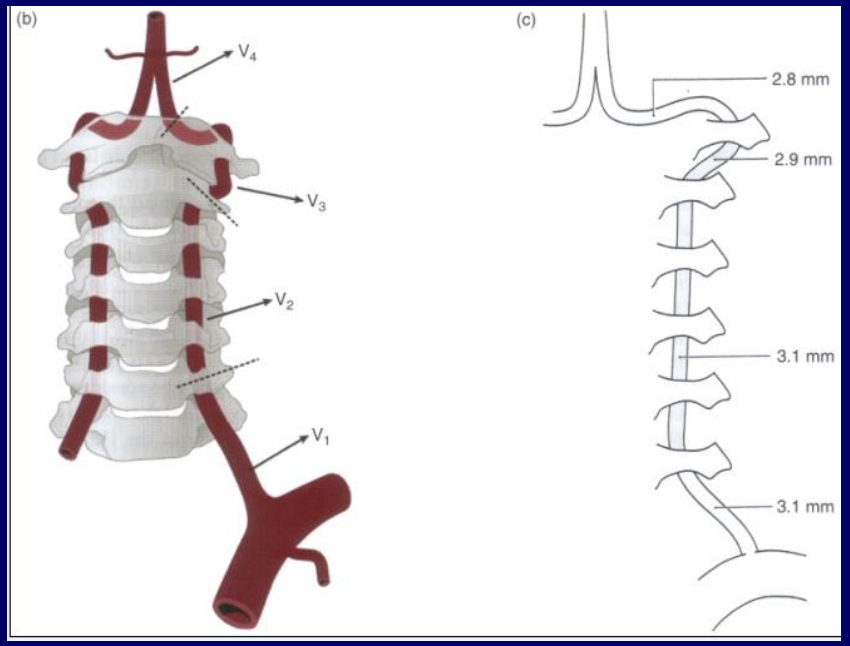
ARTERIA VERTEBRALE EXTRACRANICA

V2

CENNI ANATOMICI

Impegna il 6 forame trasverso di C 6 e termina quando fuoriesce da C2

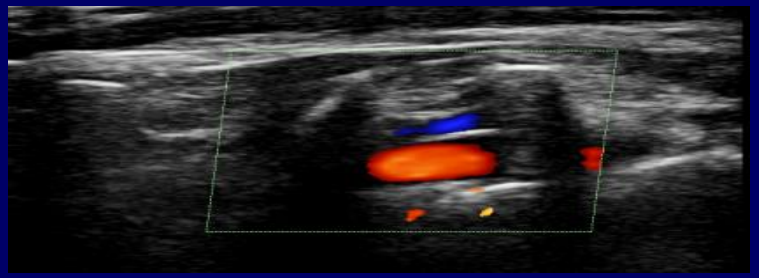




ARTERIA VERTEBRALE EXTRACRANICA

V2

IDENTIFICAZIONE



Insonazione nel 100%.

Testa del pz dritta, sonda laterale alla laringe diretta posteriormente e lievemente mediale in direzione del forame trasverso.

Iniziare in B mode per trovare il forame trasverso. Poi color e doppler.

Diametro, direzione flusso e velocità vanno documentate.

La misura del diametro deve essere attenta a non includere la vena.

Se non visualizzata iniziare con la controlaterale(Utile in ipoplasie).

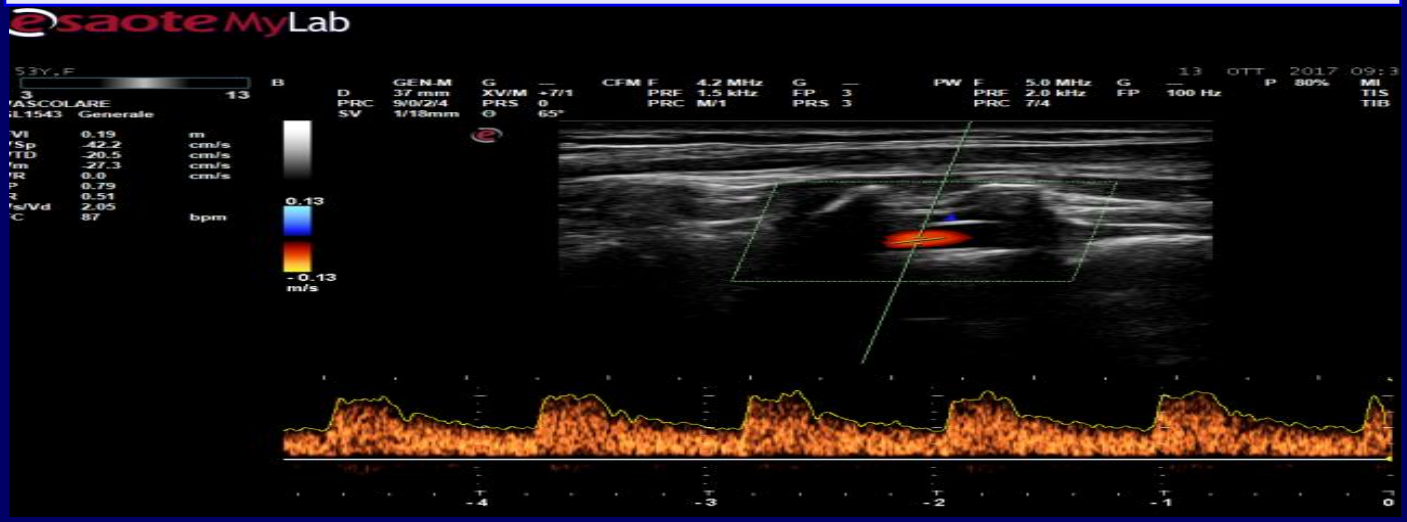
La stessa posizione della sonda sarà utilizzata controlateralmente.

ARTERIA VERTEBRALE EXTRACRANICA

V2

VALORI NORMALI lo spettro può variare da alte resistenze in ipoplasia a alti flussi diastolici in ipertrofia.

	VPS ± DS (cm/sec)	VPD± DS (cm/sec)	bibliografia
V2 dx	49±11	16±5	Kuhl 2000
V2 sx	51±10	16±4	

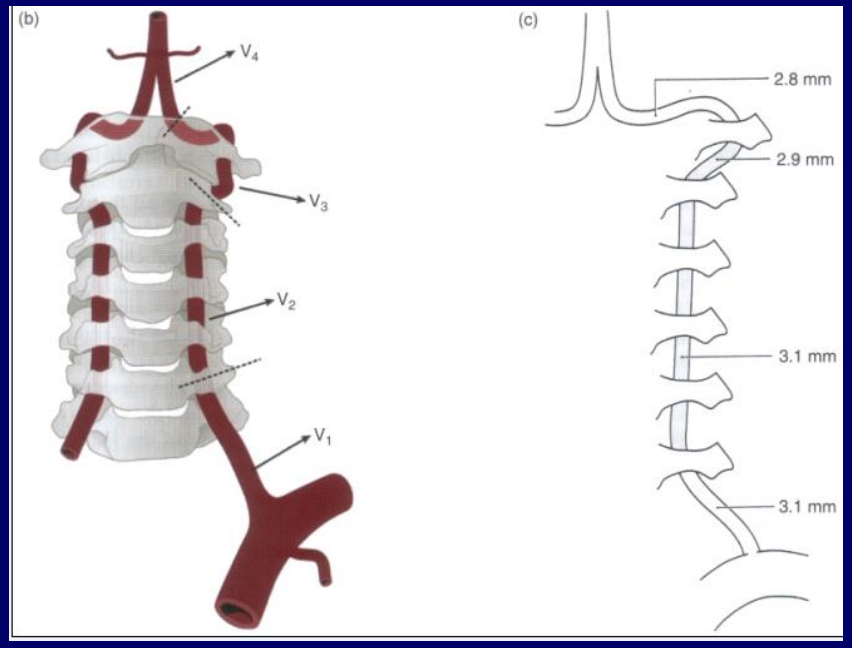


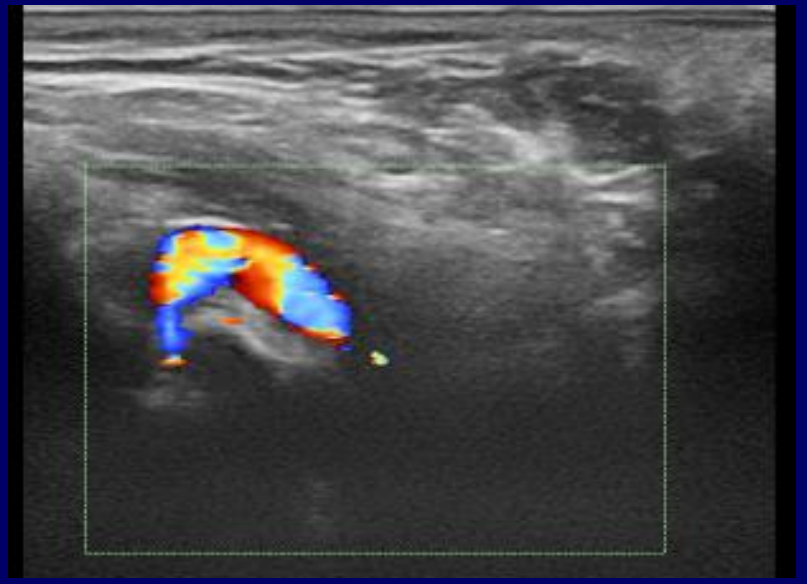
ARTERIA VERTEBRALE EXTRACRANICA

V3

CENNI ANATOMICI

fuoriesce da C2 si dirige lateralmente per poi proseguire verso l'alto fra l'epistrofeo e l'atlante con decorso verticale sino al processo trasverso dell'atlante. Esce dal forame dell'atlante e prosegue nel solco dell'arteria vertebrale . Qui originano rami che si anastomizzano con rami dell'arteria occipitale.



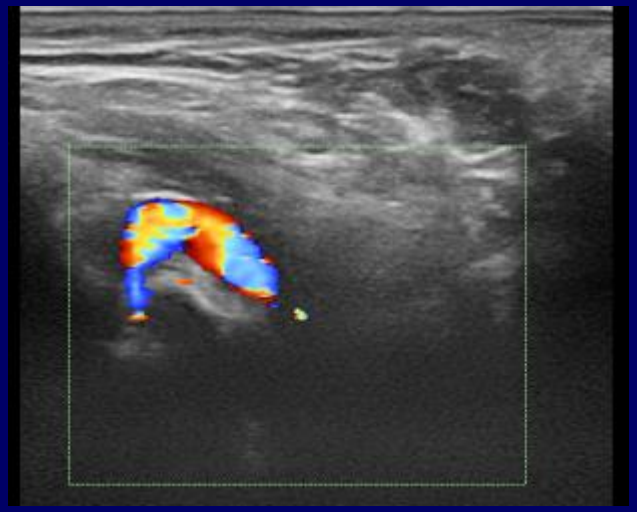


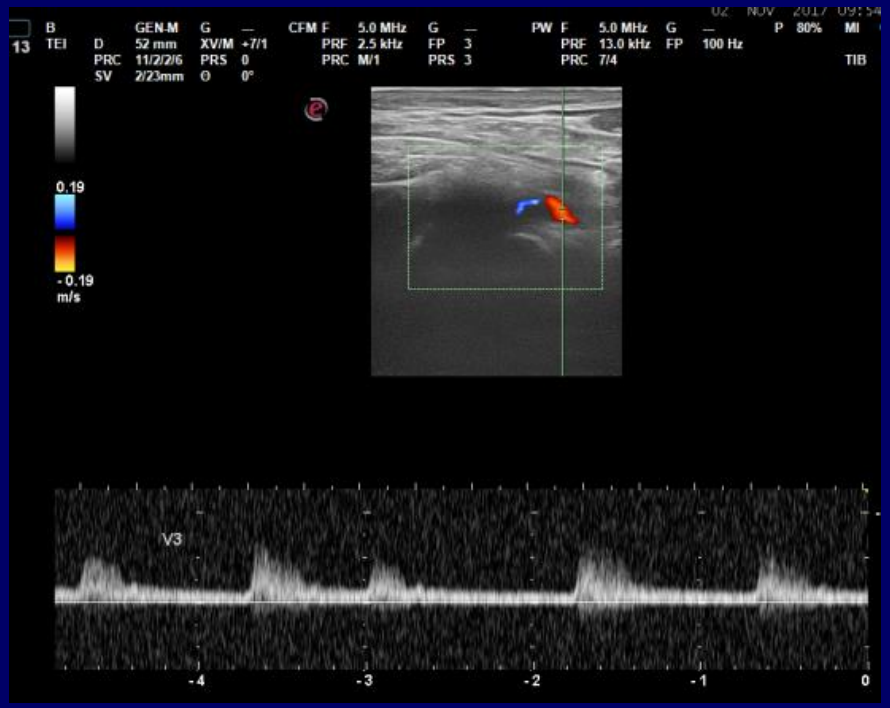
ARTERIA VERTEBRALE EXTRACRANICA

V3

IDENTIFICAZIONE VASO

Far girare la testa del paziente con sonda posta in posizione trasversa sotto il processo mastoideo.

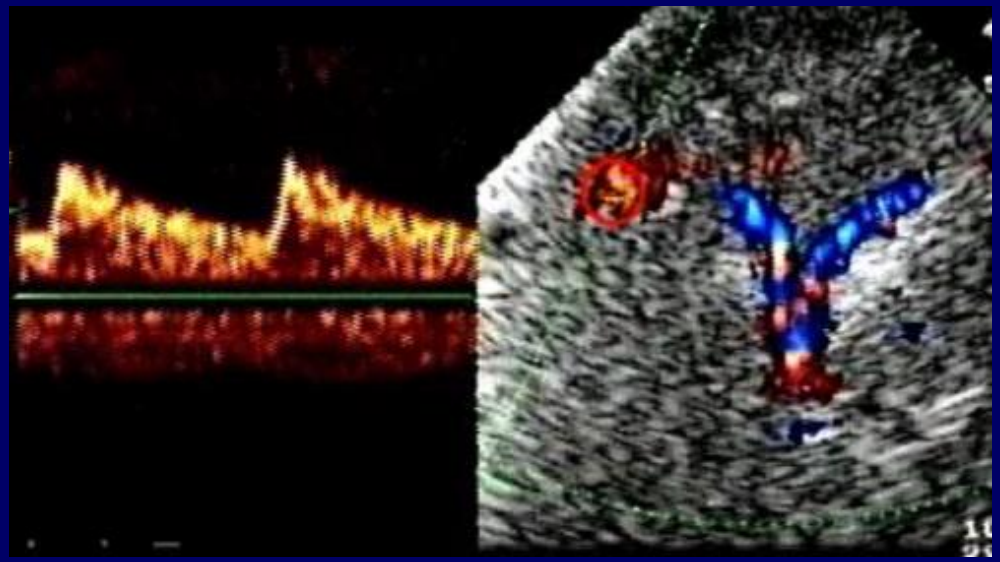




ARTERIA VERTEBRALE EXTRACRANICA

V3

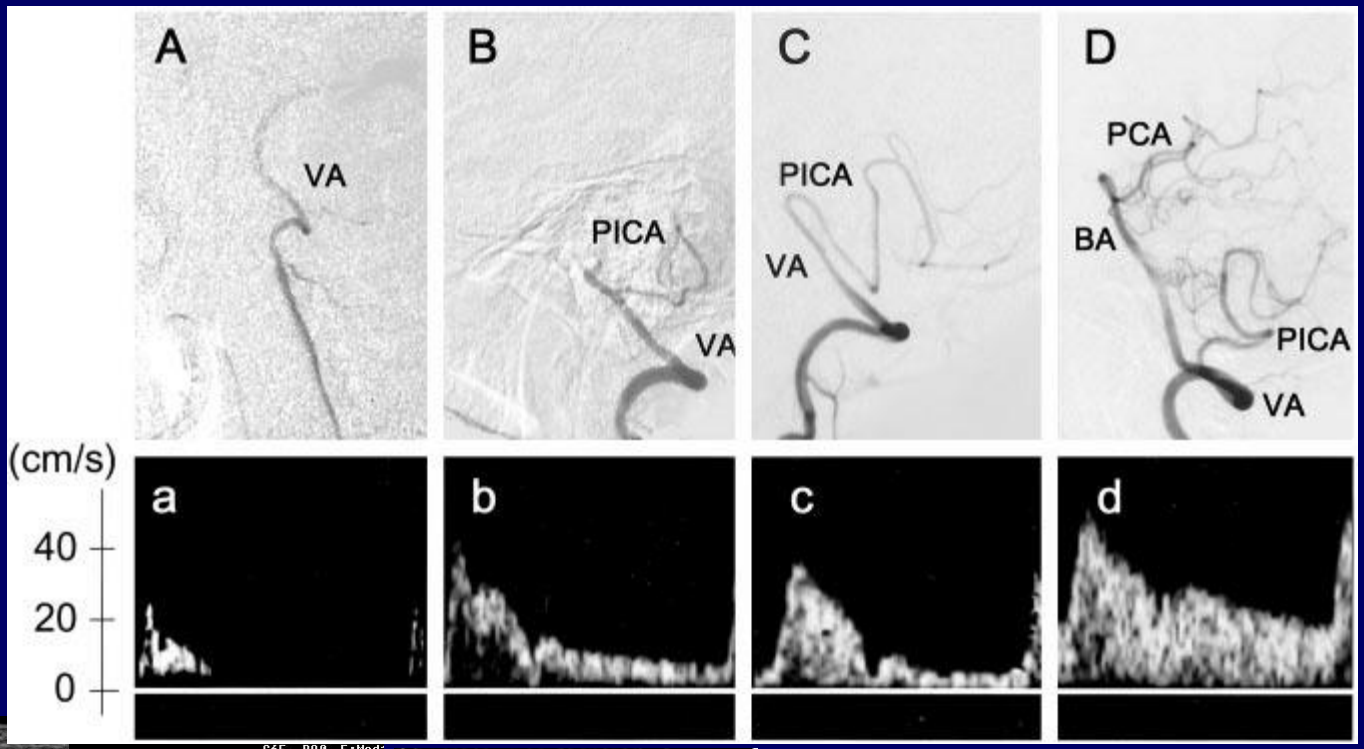
VALORI NORMALI Non ci sono valori di riferimento riportati

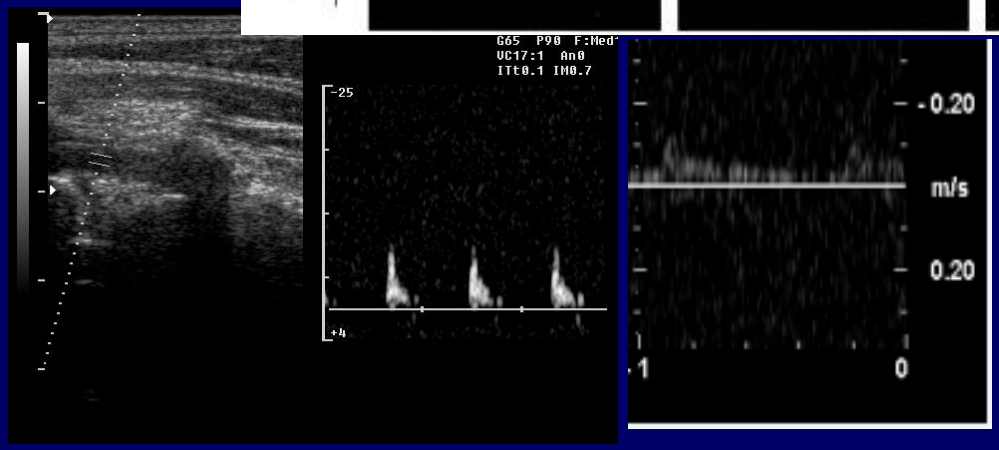


REPERTI ECODOPPLER NELLA PATOLOGIA VERTEBRALE

	DISTRETTO	VA IPSILATERALE	VA CONTROLATERALE
IPOPLASIA	V0-V4	< VD / D<2 mm	D >(ad eccezione se presenza art trigeminale), velocità normali
STENOSI	EXTRA(V0)	>V /FLUSSI POSTSTENOTICI/ COLLATERALI	>V
	INTRA	<VD EXTRA	>V
OCCLUSIONE	EXTRA V0V2	NOSEGNALE/COLLATERALI IN V2-V3	>V
	INTRA PROSSIMALE PICA	<V E NO FLUSSO DIASTOLICO IN V0-V3/ FLUSSO RETROGRADO DISTALE ALL'OCCLUSIONE PER SUPPLIRE LA PICA	>V
	INTRA DISTALE PICA	<V CON FLUSSO DIASTOLICO PRESERVATO	>V

REPERTI ECODOPPLER NELLA PATOLOGIA VERTEBRALE



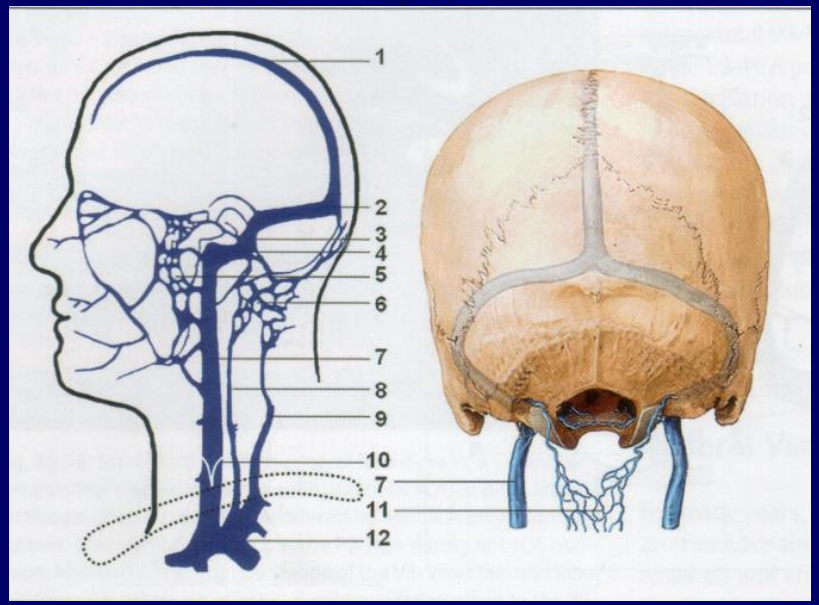


Vertebral Artery Occlusion in Duplex Color-Coded Ultrasonography

Kozue Saito, MD; Kazumi Kimura, MD; Kazuyuki Nagatsuka, MD; Keiko Nagano, MD; Kazuo Minematsu, MD; Satoshi Ueno, MD; Hiroaki Naritomi, MD

PROSPETTIVE FUTURE……

L’ OUTFLOW …



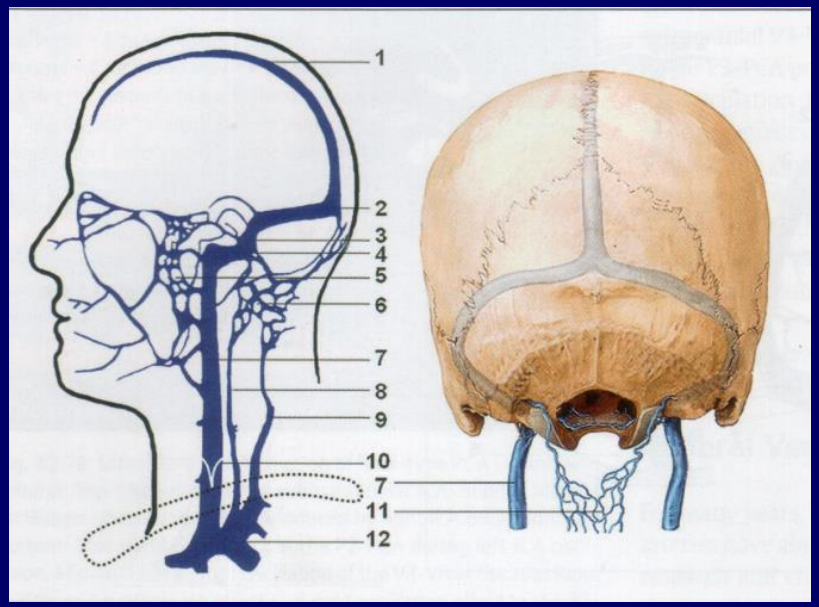
CIRCOLO VENOSO EXTRACRANICO

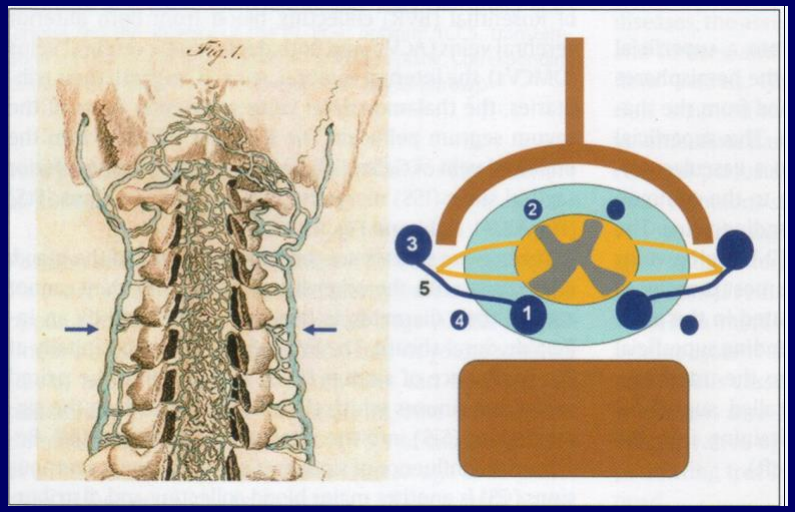
- VENA GIUGULARE INTERNA
- VENA VERTEBRALE

ANATOMIA VENOSA EXTRACRANICA

CENNI ANATOMICI

Il sistema venoso profondo e superficiale intracranico giunge nel seno petroso inferiore e nel seno sigmoideo e da qui nella giugulare .





CENNI ANATOMICI

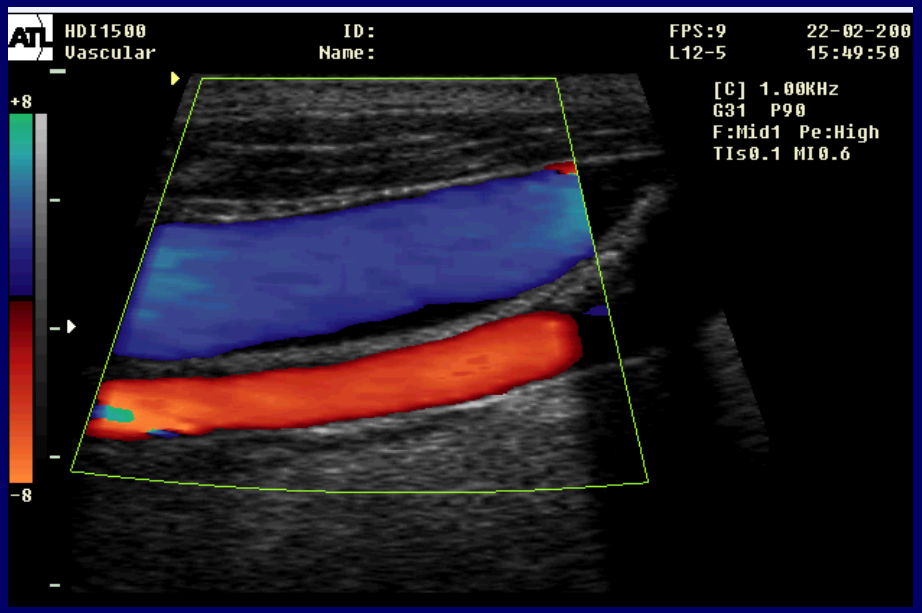
VG in clino.

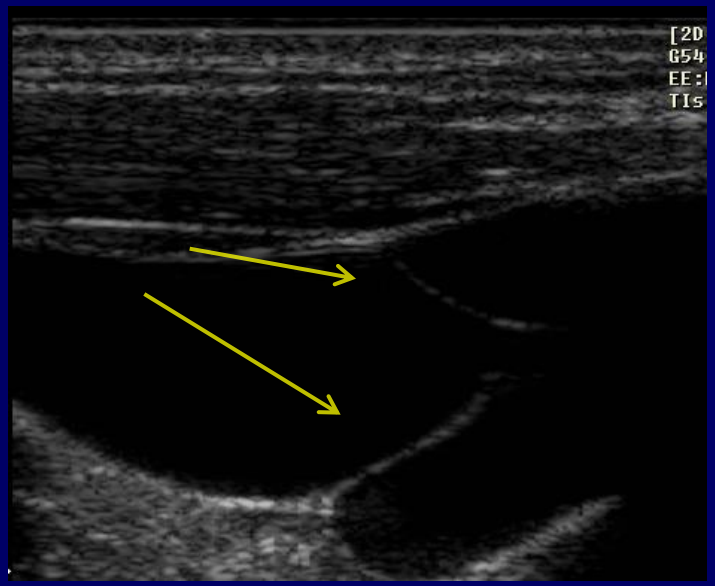
SISTEMA V VERTEBRALE (intraspinale ant e post/extraspinale ant e post) in orto perché non tendono a collabire come le VG per il rapporto con le strutture ossee

VENA GIUGULARE INTERNA

CENNI ANATOMICI

Dx dominante. Prima di terminare e con la succlavia dare la brachiocefalica, si dilata nel bulbo giug inf dove ci sono valvole .





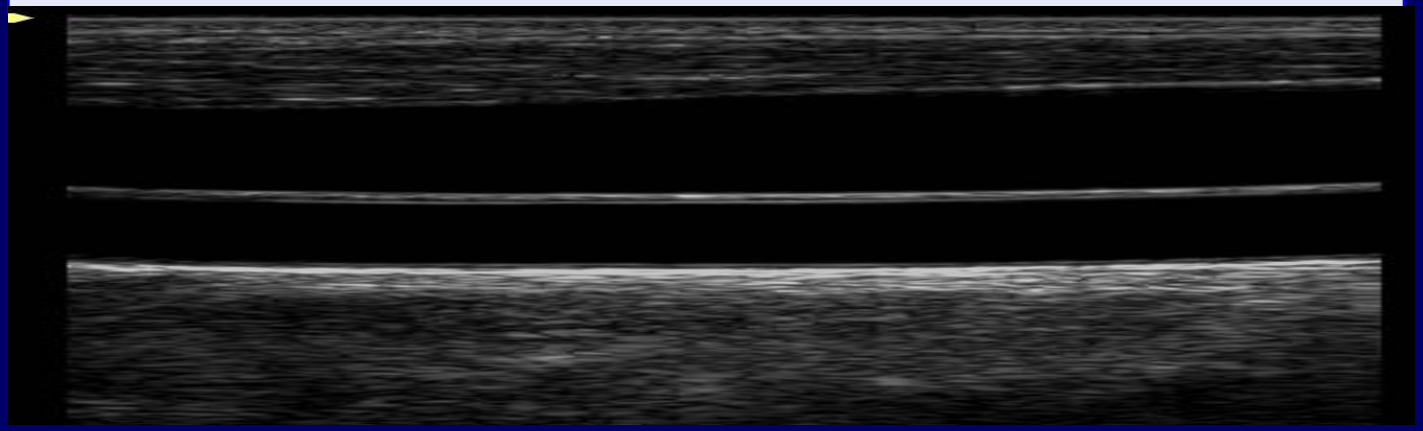
IDENTIFICAZIONE

Paziente con testa dritta per riproducibilità risultati. Piani longitudinali e trasversali. Valutare reflusso durante Valsalva. La compressione di una determinerà aumento di flusso della controlaterale se i seni trasversi sono pervi e connessi fra loro tramite la confluenza dei seni.

VENA GIUGULARE INTERNA

VALORI NORMALI molto variabili.

	VPS ± DS (cm/sec)	VPD± DS (cm/sec)	bibliografia
VGIdx VGIsx	28±15 22 ±16	21±14 18±14	Pucheu 1994



VENA VERTEBRALE

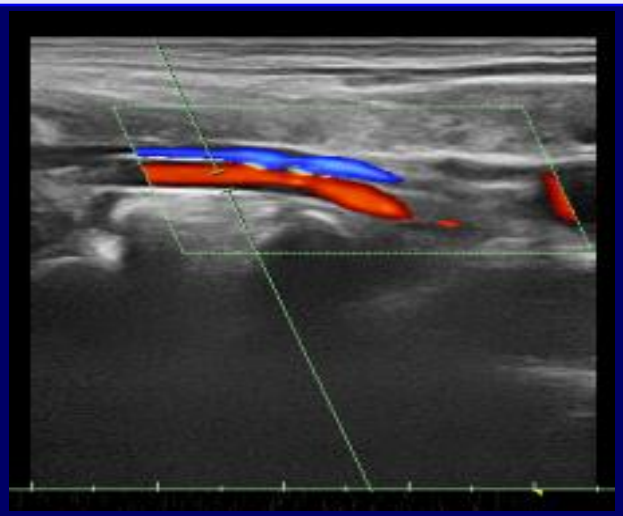
CENNI ANATOMICI

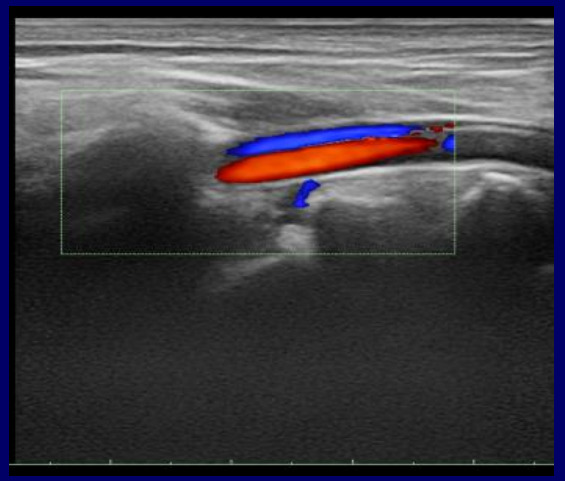
Non collassa con i cambi di posizione del capo.

IDENTIFICAZIONE

Visualizzare V2. ridurre PRF.. Compare sopra la arteria con flusso invertito. La compressione bilaterale delle giugulari determinerà aumento di flusso .

	VPS ± DS (cm/sec)	VPD± DS (cm/sec)	bibliografia
VVdx	24±12	8±8	Hoffmann 2000
VVsx	24 ±13	8±8	





TAKE HOME MESSAGE

L'esecuzione del TSA è fondamentale per approcciarsi alla patologia neurovascolare

Come tutte le metodiche per immagini ha punti di forza e debolezze

Si integra alle altre metodiche

Va considerata alla stregua del fonendoscopio per i medici che si approcciano alla patologia cerebrovascolare