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Common Carotid Artery Occlusion: Evaluation with Duplex Sonography

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PURPOSE: To demonstrate the efficacy of carotid duplex ultrasound to diagnose common carotid artery occlusion (CCAO) and to define the clinical features of CCAO. **METHODS:** We reviewed 5400 carotid duplex ultrasonograms obtained over a 7-year period for suspected carotid artery disease. In cases of CCAO, medical records were reviewed. **RESULTS:** Thirteen cases (0.24%) of CCAO were diagnosed by carotid duplex ultrasonography, including five cases of isolated CCAO. Seven cases were proved by cerebral angiography. Cerebral angiography failed to demonstrate patent internal carotid arteries in two cases of isolated CCAO. Mean age of onset was 67 ± 9 years. The main clinical presentation was stroke in nine cases (69%). The most common vascular risk factors were hypertension (62%) and heart diseases (54%). Three patients had a history of radiation therapy to the neck. Two of five patients with isolated CCAO had major stroke, with good recovery in one, whereas five of eight patients with CCAO had major stroke; among them, only one had good recovery. **CONCLUSION:** Patients with isolated CCAO may have a better outcome than patients with CCAO. Duplex sonography, particularly with color-coded flow imaging, provides an accurate examination to define the patency of the arteries distal to the carotid bifurcation. The clinical features of CCAO are similar to those of internal carotid artery occlusion except for the low prevalence of CCAO.

Index terms: Arteries, carotid, common; Arteries, stenosis and occlusion; Arteries, ultrasound

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Extracranial carotid artery disease plays an important role in the pathogenesis of hemispheric ischemic stroke. Clinical and radiologic features of internal carotid artery (ICA) occlusions have been well studied (1–5). Contrarily, common carotid artery (CCA) occlusion (CCAO) is much rarer than that of the ICA (1–5). Its clinical presentations, stroke mechanisms, treatment, and outcomes remain nebulous. CCAO is usually associated with occlusion of the ipsilateral ICA, the external carotid artery (ECA), or both. However, the ICA and ECA may be occasionally patent in cases of CCAO. Recognition of this pathologic variant enables the possibility of surgical intervention. Duplex sonography and color Doppler provide an indispensable role in detailed hemodynamic study. Herein, we report 13 cases of CCAO, including 5 cases of isolated CCAO. The clinical features, sonographic and angiographic findings, treatment, and possible mechanisms for the occlusions are discussed.

Subjects and Methods

We reviewed 5400 duplex ultrasonograms studied for detecting suspected carotid artery disease from July 1987 through June 1994. The Diasonics (Milpitas, Calif) duplex ultrasound system (DRF 400), with a transducer combining a 7.5-MHz real-time B-mode imaging and a 3.0-MHz pulsed Doppler, was used in this study. A 4.5-MHz continuous-wave Doppler was also applied to detect the flow direction in the ophthalmic artery. Echotomographies, including the CCA, ICA, and ECA with sagittal (anterioposterior, posterioanterior, and lateral) and transverse views, were carried out with the B-mode system. Two cases were demonstrated by color flow imaging with an Acuson (Mountain View, Calif) 128XP/10 and ATL (Bothell, Wash) Ultramark 9. The duplex criteria of carotid artery occlusion include: (*a*) increased echogenicity throughout the course

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TABLE 1: Stroke risk factors and clinical features in 13 patients with CCAO

Patient	Age, y/ Sex	Side of Occlusion	Risk Factors	Major Symptoms and Signs
1	79/F	L	HT, HL, stroke	Major stroke: consciousness disturbance, motor aphasia, hemiplegia
2	67/M	L	HT, HD, RT	Dizziness
3	56/M	R	HT, HD, smoking	Major stroke: consciousness disturbance, conjugate eyeball deviation, hemiplegia
4	79/M	R	HD, HL, transient ischemic attack	Amaurosis fugax
5	71/M	L	HT, HD, HL, smoking, peripheral vascular disease	Vertebral transient ischemic attack: syncope, vertigo, drop attack
6	58/M	L	HT, HD, RT, family history of stroke	Minor stroke: hemiplegia, L carotid transient ischemic attack
7	64/F	R	HT, HD, diabetes mellitus	Major stroke: consciousness disturbance, hemiplegia, hemianopsia
8	63/M	L	HT, smoking, stroke, alcohol, family history of stroke	Major stroke: consciousness disturbance, conjugate eyeball deviation, global aphasia, hemiplegia, hemianopsia
9	47/M	L	Not found	Major stroke: consciousness disturbance, conjugate eyeball deviation, motor aphasia, hemiplegia, hemianopsia
10	79/F	L	HD, HL	Major stroke: consciousness disturbance, conjugate eyeball deviation, global aphasia, hemiplegia
11	79/M	L	HD, alcohol	Minor stroke: dizziness, hemiparesis, hemihypesthesia
12	60/M	L	RT, smoking, alcohol	Syncope
13	72/M	R	HT, smoking	Major stroke: consciousness disturbance, conjugate eyeball deviation, hemiplegia, hemianopia

Note.—HT indicates hypertension; HL, hyperlipidemia; HD, heart disease; and RT, radiation therapy.

of the vessel; (*b*) a lack of cross-sectional pulsation; and (*c*) an absence of flow signal.

The cerebral angiograms were obtained from a digital subtraction angiography unit. The contrast medium used was 65% meglumine diatrizoate with a 2:1 dilution in normal saline. The injection rate was 15 to 20 mL/s for the aortic arch, 8 to 10 mL/s for the subclavian artery or brachiocephalic artery, 6 to 8 mL/s for selective CCA study, and 3 to 5 mL/s for the vertebral artery. The total doses injected were 30 to 40, 14 to 20, 8 to 10, and 5 to 7 mL for the aortic arch, subclavian artery or brachiocephalic artery, CCA, and vertebral artery, respectively. The serial films for each patient are two films per second for the first two seconds, then one film per second for the following 18 seconds to ensure adequate delayed film for collateral circulation.

The clinical features, risk factors, and history were obtained from detailed review of medical records. Computed tomography (CT) and angiography were interpreted by two neurologists and one neuroradiologist. Brain CT was done in all patients with CCAO. One patient also had a neck CT. CCAO was further proved by cerebral angiography in seven patients. Only one patient received surgical treatment with a subclavian artery–ECA and superficial temporal artery–middle cerebral artery bypass procedure.

Cardiac diseases defined as risk factors of cerebrovascular disease are: angina, myocardial infarction, nonphysiologic arrhythmia, mitral or aortic valvular stenosis or regurgitation, cardiomegaly, left ventricular hypertrophy, and left atrial enlargement. Major stroke was defined as a combination of consciousness disturbance and at least two of the following neurologic signs: conjugate deviation, homonymous hemianopsia, aphasia, and dense hemiplegia. Others were classified as minor stroke.

Results

Thirteen cases fulfilled the criteria of CCAO, representing 0.24% of all patients who underwent carotid duplex examination during the 7-year period. Among these cases, only five patients had patent ICAs and ECAs. Meanwhile, 137 cases of ICA occlusion (2.5%) were also found from the same data bank.

Table 1 shows the clinical features and outcomes of the 13 patients with CCAO. The mean age of onset was 67 $\pm$ 9 years (range, 47 to 49 years). Ten (77%) were men and 3 were women. Five of the 13 patients had isolated CCAO (patients 1 through 5). Occlusion was predominant on the left (9 of 13 patients, 69%). The most prevalent risk factors were hypertension (8 patients, 62%) and cardiac disease (7 patients, 54%). Other risk factors included cigarette smoking (5 patients), hyperlipidemia (4 patients), previous stroke (2 patients) and transient ischemic attack (1 patient), alcohol consumption (3 patients), history of radiation therapy to the neck (3 patients), diabetes mellitus (1 patient), and peripheral vascular disease (1 patient). Twelve patients (92%) had more than

TABLE 2: Findings of Doppler ultrasonography, brain CT, and cerebral angiography in 13 patients with CCAO

Patient	Side of Occlusion	Doppler Ultrasound			Brain CT	Cerebral Angiography	Outcome
		ICA	ECA	OA			
1	L	An	Re	Re	Multiple small infarcts	. . .	Partial dependence, recurrent stroke
2	L	An	Re	An	Cortical atrophy	L CCA occlusion, delayed filling of L ICA and ECA via L subclavian artery	No deficit
3	R	An	Re	An	R MCA territory infarct	Occlusions of L CCA and ICA	Partial dependence
4	R	An	Re	An	Cortical atrophy	R CCA occlusion, delayed filling of R ICA and ECA via R extracranial vertebral artery	No deficit
5	L	Re	An	Re	Normal	Occlusion of L CCA and R ICA, filling of L ICA via L posterior communicating artery	No deficit
6	L	−	−	Re	Normal	. . .	Total dependence, recurrent transient ischemic attack and stroke
7	R	−	−	Re	R MCA territory infarct	Occlusion of R CCA, ICA, ECA, and R posterior cerebral artery	Partial dependence
8	L	−	−	Re	L MCA territory infarct	. . .	Total dependence
9	L	−	−	Re	L MCA territory infarct	Occlusion of L CCA, ICA, ECA, and L subclavian artery	Recurrent stroke and death
10	L	−	−	Re	L MCA territory infarct	. . .	Total dependence
11	L	−	−	Re	Cortical atrophy	. . .	Partial dependence, recurrent stroke
12	L	−	−	Re	Normal	Occlusion of L CCA, ICA, ECA	No deficit
13	R	−	An	Re	R MCA territory infarct	. . .	Total dependence

Note.—An indicates antegrade; Re, retrograde; −, no flow; MCA, middle cerebral artery; and OA, ophthalmic artery.

two vascular risk factors. Eleven patients (85%) presented with strokes or transient ischemic attacks, ipsilateral to the lesion side. Only 1 patient (patient 6) had a bilateral presentation. Seven of them suffered major strokes. Three patients had carotid transient ischemic attacks. One patient (patient 5) presented with syncope, drop attack, and vertigo, corresponding to vertebrobasilar transient ischemic attack. The only asymptomatic patient (patient 2) felt dizzy before examination. All patients but 1 (patient 3) received medical treatment, including aspirin, warfarin, or heparin. Four cases had episodes of recurrent stroke. Most of the victims of major stroke had sequelae of neurologic deficits with dependent daily life. Among the 5 patients with isolated CCAO, patient 3 underwent an operation of subclavian-ECA and superficial temporal artery–middle cerebral artery bypass and had good functional recovery. Another 4 cases had no or little sequelae with medical treatment.

Table 2 outlines the findings of the ultrasonography, brain CT, and cerebral angiography in 13 patients with CCAO. Results of Doppler study showed antegrade flow in the ipsilateral ICA and retrograde flow in the ipsilateral ECA in 4 patients (patients 1 to 4). Three of them had antegrade ophthalmic flow, and 1 had retrograde flow. Patient 5 had retrograde ipsilateral ICA flow, antegrade ipsilateral ECA flow, and retrograde ophthalmic flow. One patient (patient 3) with isolated CCAO is illustrated in Figure 1A. Color flow Doppler was applied in patients 2 and 13. In patient 2, color flow Doppler imaging clearly showed neither color nor flow in the occluded left CCA, and the distal blood flow was supplied from the left ECA with drainage into the left ICA (Fig 2A). In patient 13, color flow Doppler displayed occlusion throughout the right CCA to the ICA and proximal ECA. Antegrade flow was detected in the right ECA distal to the branch of superior thyroid artery, which served as a collateral from the contralateral carotid artery. This disappeared during transient compression of the contralateral CCA (Fig 3). The other patients (patients 6 to 12) had complete occlusion of the CCA, ICA, and ECA with retrograde ophthalmic flow.

The brain CT in patients having had major stroke (patients 1, 3, 7 through 10, and 13)

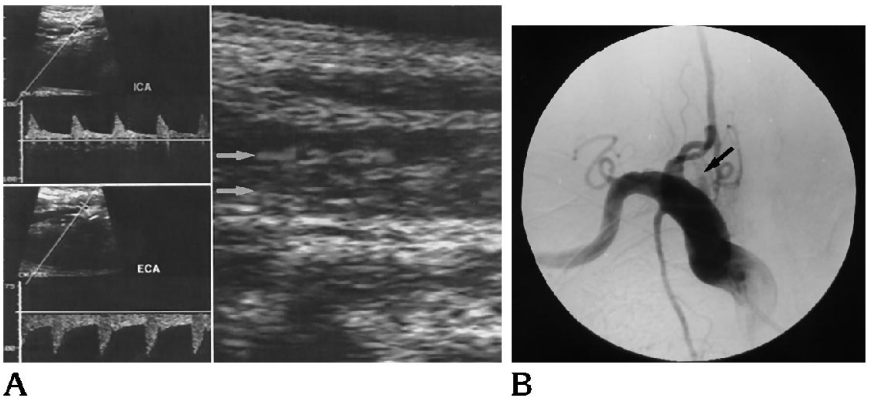

Fig 1. Patient 3. CCAO with patent ICA and ECA.

A, Carotid duplex sonography shows hyperechogenicity filling in the whole lumen throughout the right CCA (*arrows*). Doppler studies demonstrate retrograde flow in the right ECA (*left lower*) and antegrade flow in the right ICA (*left upper*).

B, Angiogram from right brachiocephalic artery injection shows nonopacification of the right CCA (*arrowhead*).

showed either large or multiple small hypodense lesions in the ipsilateral hemisphere. The CT findings of the others (patients 2, 4, 5, 6, 11, and 12) were essentially normal or displayed mild cortical atrophy only. In patient 2, the CCAO was initially diagnosed by a routine follow-up neck CT for nasopharyngeal cancer, which revealed a thrombus in the left CCA (Fig 2B). Seven patients underwent cerebral angiography. Two patients (patients 2 and 4) had CCAO at their origin and delayed filling of the ICA and ECA via the subclavian and vertebral arteries, respectively (Figs 2C–F). Another two patients (patients 3 and 5) had left CCAO with no visibility of the left ICA on angiography (Fig 1B). However, the left ICA was demonstrated to be patent by ultrasonography (Fig 1A). In patients 7, 9, and 12, who were diagnosed as having complete occlusion of the CCA, ICA, and ECA by ultrasound, the angiography showed similar results.

Discussion

The prevalence of CCAO in patients with symptomatic cerebrovascular disease has been reported in 1% to 5% of cases (5–7). From our neurosonology data bank, ICA occlusion was found in 2.5% and CCAO in only 0.24%. The ratio of CCAO to ICA occlusion was around 1 of 10, which is similar to that of angiographic series (5). Isolated CCAO was found to affect only 0.1% of our patients; however, the actual prevalence of CCAO is likely to be underestimated, because some patients may be asymptomatic. Most of the patients are male (6–9). CCAO had a higher prevalence of left side involvement in our study and in previous series (5, 6, 9, 10). This side-to-side difference may be attributable to varying flow dynamics and arterial length differences between either sides. The left CCA usually has a more acute angular takeoff from the aorta than the right (11). Most of the patients with CCAO were symptomatic, presenting with transient ischemic attacks or strokes ipsilateral to the lesion side. In our series, the occurrence of stroke was more common than transient ischemic attack, and the majority were major strokes. Such presentations were different from reported series in which transient ischemic attacks were predominant (7–9, 12).

The most important cause of CCAO is atherosclerosis. Other major causes include arteritis such as Takayasu disease, fibromuscular dysplasia, thrombocytosis, dissection of the CCA or the aortic arch, aortic arch aneurysm, iatrogenic occlusion, mediastinal tumors, cardiac embolism, postirradiation arteropathy, blunt or open craniocervical trauma and idiopathic occlusion (1, 12). Collice et al showed that hypertension and heart disease with abnormal electrocardiograms were the major risk factors (6). In our series, 92% of the patients had two or more risk factors; among these, hypertension (62%) and heart diseases (54%) were the most prevalent. Radiation therapy to the neck region also plays an important role in the pathogenesis of CCAO; although this was rarely reported in other series. High-dose cervical irradiation for cancer in the neck and head regions may accelerate carotid atherosclerosis.

The conventional diagnostic procedure for carotid artery diseases is angiography; however, in cases of CCAO it has limitations in the evaluation of the carotid artery distal to the occlusion. The limitations of cerebral angiography might be attributable to inadequate concentration of contrast medium and trivial flow in the major branches, variable collateral circulation, and poor delayed angiograms in most studies

Fig 2. Patient 2. CCAO with patent ICA and ECA.

A, Color flow Doppler imaging of the left carotid bifurcation shows a hyperechogenic lesion in the lumen of the CCA (*arrow*) and retrograde flow (*orange*, toward the transducer) in the ECA with reconstruction of antegrade flow in the ICA (*blue*, away from the transducer).

B, Neck CT shows a filling defect after contrast enhancement in the left CCA with homogeneous material in the lumen (*large arrow*). The right CCA was well opacified with contrast media (*small arrow*). *C*, Angiography demonstrates left CCAO at the origin (*arrow*).

D–F, Lateral view of sequential angiograms from left subclavian artery injection. Initially, faint opacification of the left ECA (*small arrows*) fills from a branch (*large arrowheads*) of the left subclavian artery and the superior thyroid artery (*small arrowheads*) (*D*). The following film showed more opacification of the left ECA (*small arrows*) and ICA (*large arrow*) (*E*). Finally, opacification of the left ICA (*large arrow*) and ECA (*small arrows*) is demonstrated in the delayed film (*F*).

Fig 3. Patient 13. CCA and ICA occlusion with patent ECA. Color flow duplex sonogram of the right carotid system. *Upper left* (sagittal view), hyperechogenic lesion in the lumen of bifurcation and proximal portion of the ECA. Antegrade flow (*blue*, away from the transducer) is noted distal to the occlusion. *Upper right* (transverse view), retrograde flow (*red*, toward the transducer) is detected in the right superior thyroid artery (*STA*) (*arrowhead*), which then fills into the right ECA. The flow in the right superior thyroid artery disappears during compression of the left CCA and appears after releasing the pressure (*lower half*).

(7, 8, 10). Nevertheless, these limitations can be improved by injecting adequate contrast medium and by obtaining delayed films to demonstrate collateral flow and delayed filling of the ICA and ECA. Podore et al reported 6 cases of successful revascularization in 12 patients, but only 2 cases with patent ICAs were demonstrated by angiography (7). Riles et al showed that the accuracy of preoperative angiographic diagnosis of patent ICA was 17% (4 of 24) (8). The addition of duplex ultrasonography has further advanced our ability to define the carotid anatomy more accurately (13, 14). Duplex ultrasonography provides good B-mode imaging of the morphology of the extracranial carotid arteries and Doppler study of the hemodynamics of the blood flows (15, 16). The overall accuracy of duplex ultrasound diagnosis of occlusion of the extracranial carotid artery was greater than 96% in our laboratory and others (17, 18). The accuracy of duplex sonography should be much higher in the diagnosis of CCAO than ICA occlusion, because of the easy accessibility to the CCA compared with the ICA. Using the color Doppler with lower pulse repetition frequency allows easy identification of the relatively low flow in the ICA and ECA distal to a CCAO, thereby excluding true occlusion. Therefore, carotid duplex ultrasound is a good guide before angiographic study for extracranial carotid artery disease.

In our patients with isolated CCAO, four had retrograde flow in the ECA and antegrade flow in the ICA (Fig 2A). This is similar to previously reported cases (9, 13). Most of the blood supply to the patent portion distal to the occluded CCA is from the extracranial arteries, such as the thyrocervical or costocervical trunk of the subclavian artery, the vertebral artery, or the contralateral ECA. These collaterals fill the ECA, resulting in retrograde flow in the ECA, and then keeping the ICA patent with antegrade flow. Collaterals from intracranial vessels via the anterior or posterior communicating arteries are rarely seen and may have retrograde flow within the ICA, such as patient 5 in our series (Table 2). The direction of ophthalmic flow is also a good indicator of the collateral circulation. Retrograde ophthalmic flow suggests an extracranial collateral. Only a patent ICA results in antegrade ophthalmic flow in our series, although there were a few reported cases with ICA occlusion and antegrade ophthalmic flow that came from middle meningeal artery (19).

Among five patients with isolated CCAO, two (40%) had minimal neurologic deficits. In all the patients with CCAO and ICA occlusion, the flow in the ipsilateral ophthalmic artery was retrograde (Table 2). Seven patients (seven of eight, 88%) had sequelae of neurologic deficits, and five of these patients had totally dependent daily lives (Table 1). We conclude that patients with isolated CCAO may have better outcomes. Patients with ICA occlusion and retrograde ophthalmic flow usually had worse prognoses with

some neurologic deficits and dependent daily lives.

The treatment of CCAO depends on the patency of the distal carotid arteries (8). To decide between an endarterectomy or an arterial bypass, recognition of patent vessels distal to the CCAO is most important. The main purpose for surgical reconstruction is to prevent stroke. Podore et al considered those who had minimal neurologic deficits from previous strokes as the candidates for cerebral revascularization (7). Surgical interventions, mainly bypass procedures, including subclavian to the CCA, subclavian to the ICA, subclavian to the ECA, axillary to the CCA, or ascending aortic bifurcation graft to the CCA, may have a higher surgical risk than endarterectomy. Whether these aggressive operations are indicated in cases of isolated CCAO is still controversial (14).

In conclusion, CCAO is unusual, and isolated CCAO is rarer. The clinical course, pathophysiology, hemodynamics, and appropriate treatment are still not well documented. Duplex sonography, especially color-coded flow imaging, provides an accurate examination of the distal carotid arteries. Patients with isolated CCAO may have better outcomes than patients who have CCAO with ICA occlusion.

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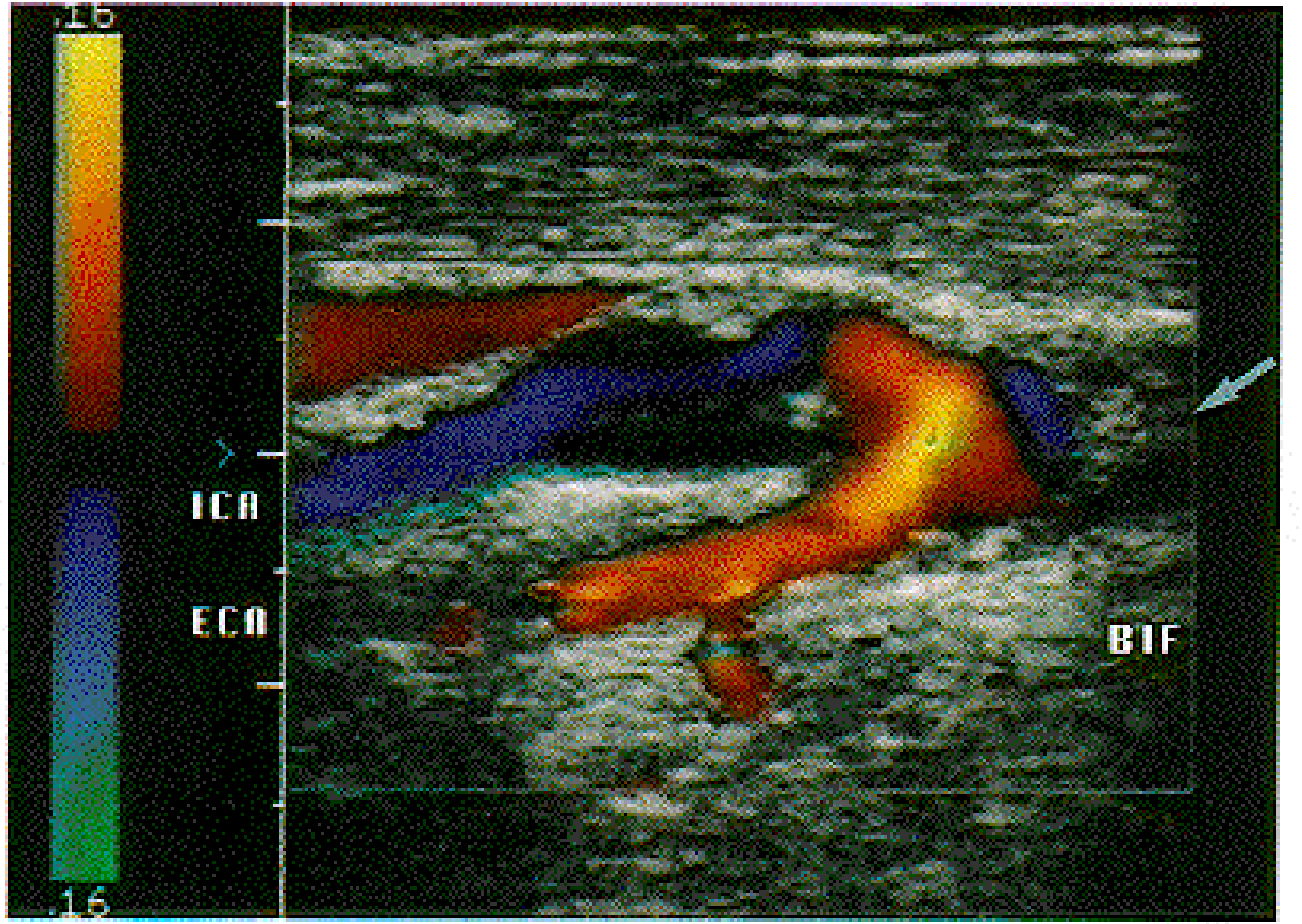

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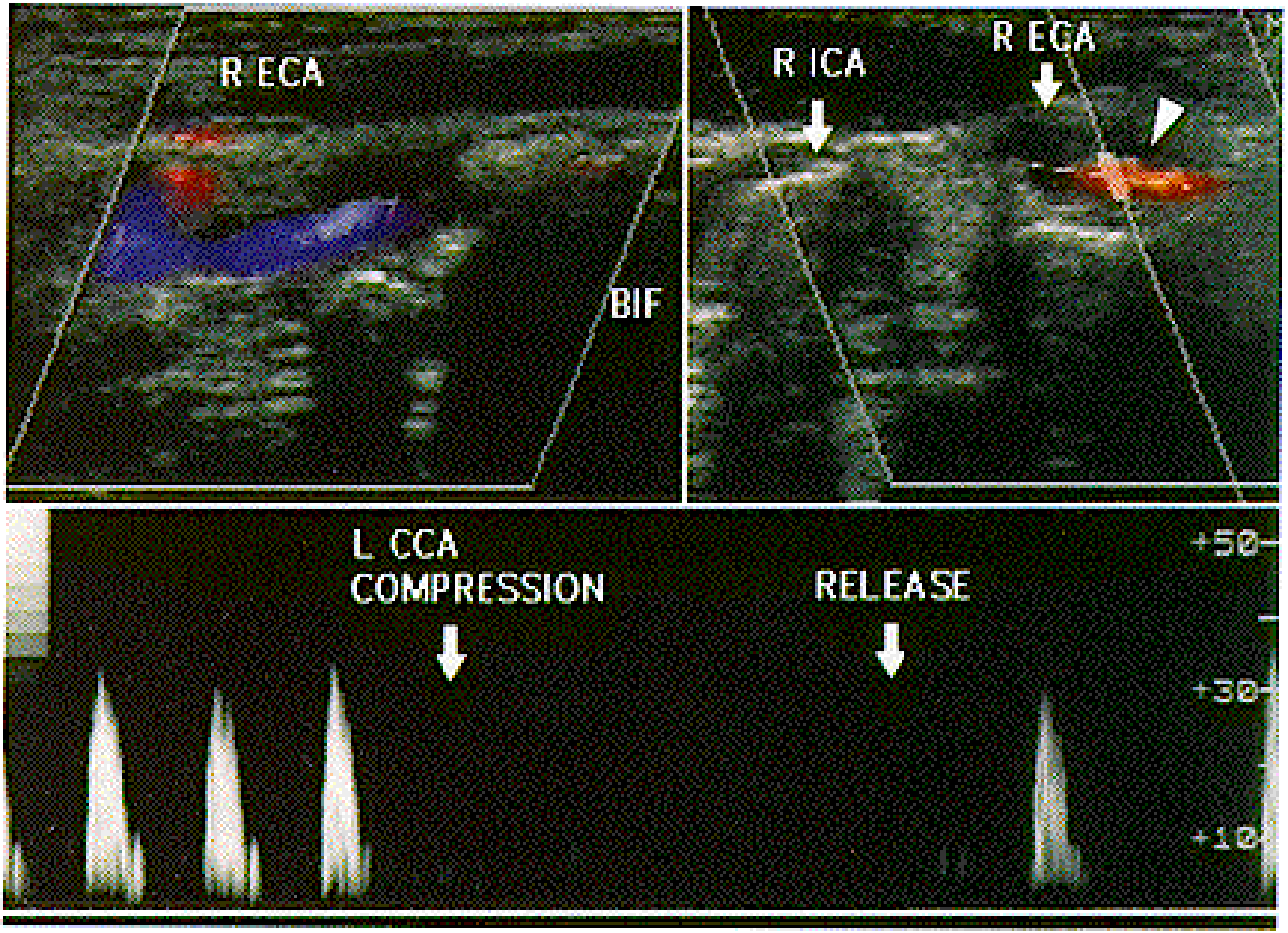
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