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Delayed reversal of vertebral artery blood flow following percutaneous transluminal angioplasty for subclavian steal syndrome

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Summary. Twelve patients suffering from subclavian steal syndrome of various severity due to either proximal subclavian stenoses (10 cases) or subclavian occlusion (2 cases) were treated with percutaneous transluminal angioplasty (PTA). Olbert's dilatation catheters were used. The occlusions could neither be recanalized by the transfemoral nor transaxillary approach. In one right-sided subclavian stenosis an additional distal subclavian occlusion prevented proper placement of the catheter. All the other patients were treated successfully with no re-occlusion during a follow-up period of 1 to 7 months. In one patient, a transient embolic occlusion of the finger arteries was seen following post-interventional repeat angiography. Before, during and after PTA, continuous ultrasound monitoring of the homolateral vertebral flow patterns revealed an unexpected "delay" phenomenon. Despite sufficient recanalization of the proximal subclavian artery, the flow direction within the vertebral artery did not immediately change to antegrade but rather did so gradually within 20 s up to several minutes. This delay of flow-reversal is thought to serve as a protective mechanism against cerebral embolism during, and shortly after PTA of the subclavian artery. Relying on Doppler ultrasound findings, a staging of the subclavian steal is proposed in order to allow adequate selection of patients for PTA.

Key words: Subclavian steal syndrome – percutaneous transluminal angioplasty – supraaortic vessels – delayed reversal of vertebral blood flow – Doppler sonographic monitoring – cerebral embolism

In recent years, dilating and occluding techniques during interventional neuroradiology have provided new and promising approaches to the treatment of cerebro-vascular disease and intracranial vascular malformations.

Percutaneous transluminal angioplasty (PTA) of the ileofemoral, renal and coronary arteries has already proven to be beneficial for patients suffering from occlusive arterial disease [1–3]. A short, yet comprehensive review concerning "newer applications" of PTA was published by Novelline [4] who stated, "We are just now beginning to realize the enormous potential for transluminal angioplasty in treating stenotic and occlusive lesions throughout the vascular system" (p 983).

However, clinicans have traditionally hesitated to apply transluminal angioplasty to the supraaortic, brain-supplying vessels. The main reason for this reservation was the fear of cerebral embolism during maneuvers within the brachiocephalic arteries. Consequently, only few authors have attempted to dilate the orifices of the vertebral arteries [5–8], the "string of beads" due to fibromuscular disease within the internal carotid arteries [9–12] and other segments of the carotid system [5, 13–16].

As fas as we know, Bachmann et al. [17] were the first to apply PTA to the *proximal* part of the subclavian artery in order to relieve a patient of the symptoms of his subclavian steal. Mathias et al. were able to demonstrate good results of PTA in a larger group of patients suffering from subclavian steal syndrome [7, 18, 19]. In our department, 12 patients have undergone PTA of the proximal subclavian artery. The alterations of the blood flow within the homolateral "stealing" vertebral artery were continuously registered with the help of Doppler ultrasound before, during and shortly after balloon inflation. Our findings shed some additional light on the pathophysiological events occuring during, as well as after, subclavian PTA and seem to be of relevance for its requirement and planning.

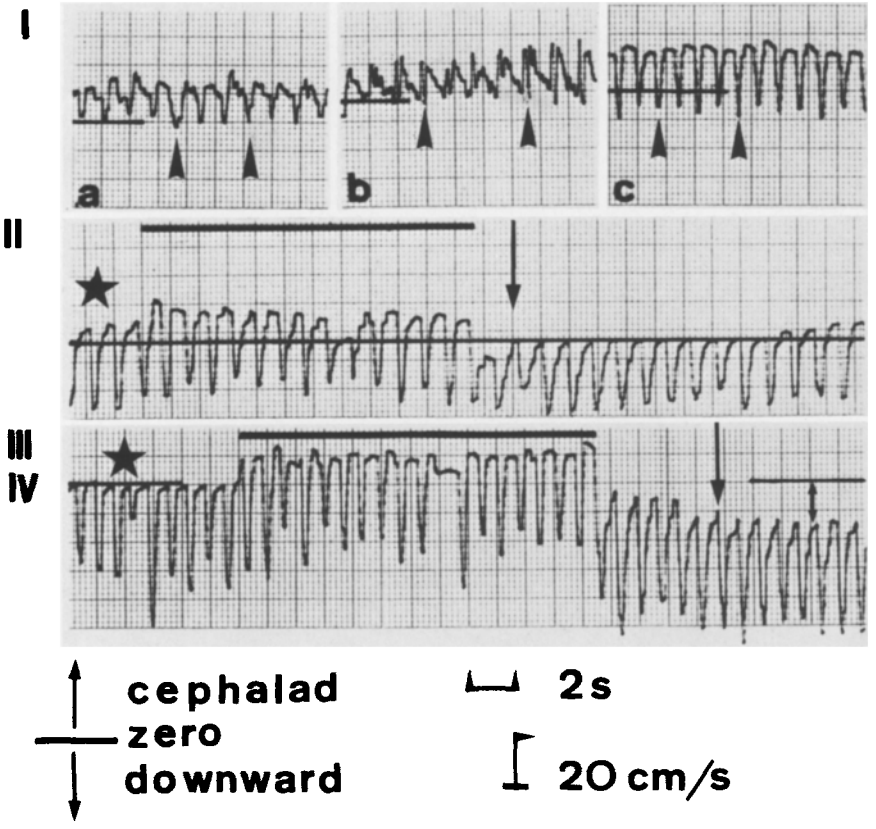

Fig. 1 a–c. Staging I–IV of subclavian steal syndrome according to Doppler sonographic findings. *I:* Cephalad blood flow is more (**b**) or less (**a**) decelerated *(arrowheads)* during early systole. (**c**) During hyperemia of the homolateral arm following brachial artery compression, an intermittent caudal blood flow may occur *(arrowheads)* but net flow is still cephalad. *II:* Predominantly caudally directed, to-and-fro flow *(asterisk)*. Lack of net movement of the blood column occurs during compression of the brachial artery *(black bar at the top)*. Hyperemia of the arm immediately leads to a "manifest" subclavian steal *(arrow)* with gradual return to the initial level. *III and IV:* Manifest steal *(asterisk)* changes to to-and-fro flow (or is at least diminished) during brachial compression *(black bar at the top)*. Hyperemia induces marked increase of flow within the stealing vertebral artery *(arrow)*. (Note high flow velocity during diastole) *(double-arrow)*

Methods

Pre-interventional diagnostics and staging of the severity of the subclavian steal

Relying on ultrasound findings, all patients presenting with subclavian steal syndrome were ranked according to the degree of the subclavian lesion and to the severity of the steal effect. At least four stages of the subclavian steal syndrome should be differentiated with respect to the hemodynamic effect of the subclavian lesion on vertebral blood flow.

Stage I: A 60–70% subclavian stenosis, as a rule, induces to-and-fro flow within the homolateral vertebral artery with predominantly cephalad blood flow. Partial reversal of the blood flow is regularly provoked by reactive hyperemia of the arm following brachial artery compression (Fig. 1, I).

Stage II: An approx. eighty percent stenosis of the proximal subclavian artery also leads to a to-and-fro movement of the vertebral blood column. The blood flow, however, is predominantly caudal. During hyperemia of the ipsilateral arm, the steal becomes manifest (Fig. 1, II).

Stage 1 and 2 may be characterized as a "latent" subclavian steal.

Stage III: More than a ninety percent stenosis of the proximal subclavian artery leads to a "manifest" subclavian steal syndrome. Even without provocative maneuvres, flow direction within the homolateral vertebral artery is completely and continuously directed downwards (Fig. 1, III).

Stage IV: The steal is also "manifest", as in stage III, but the underlying lesion is a complete occlusion of the subclavian artery.

Only stage III and IV of a subclavian steal syndrome can regularly be documented by arch aortography, whereas stage I and II can be reliably detected only with the help of vertebral Doppler sonography [20, 21].

All patients underwent thorough *clinical examination including history taking* with special reference to transient or definite ischemic brain dysfunction and/or arm claudication (see clinical data in Table 1). Additionally, all subjects were screened for clinically silent brain stem lesions by recording somatosensory and acoustically evoked potentials as well as the orbicularis oculi reflex (These data have been published elsewhere; [22]).

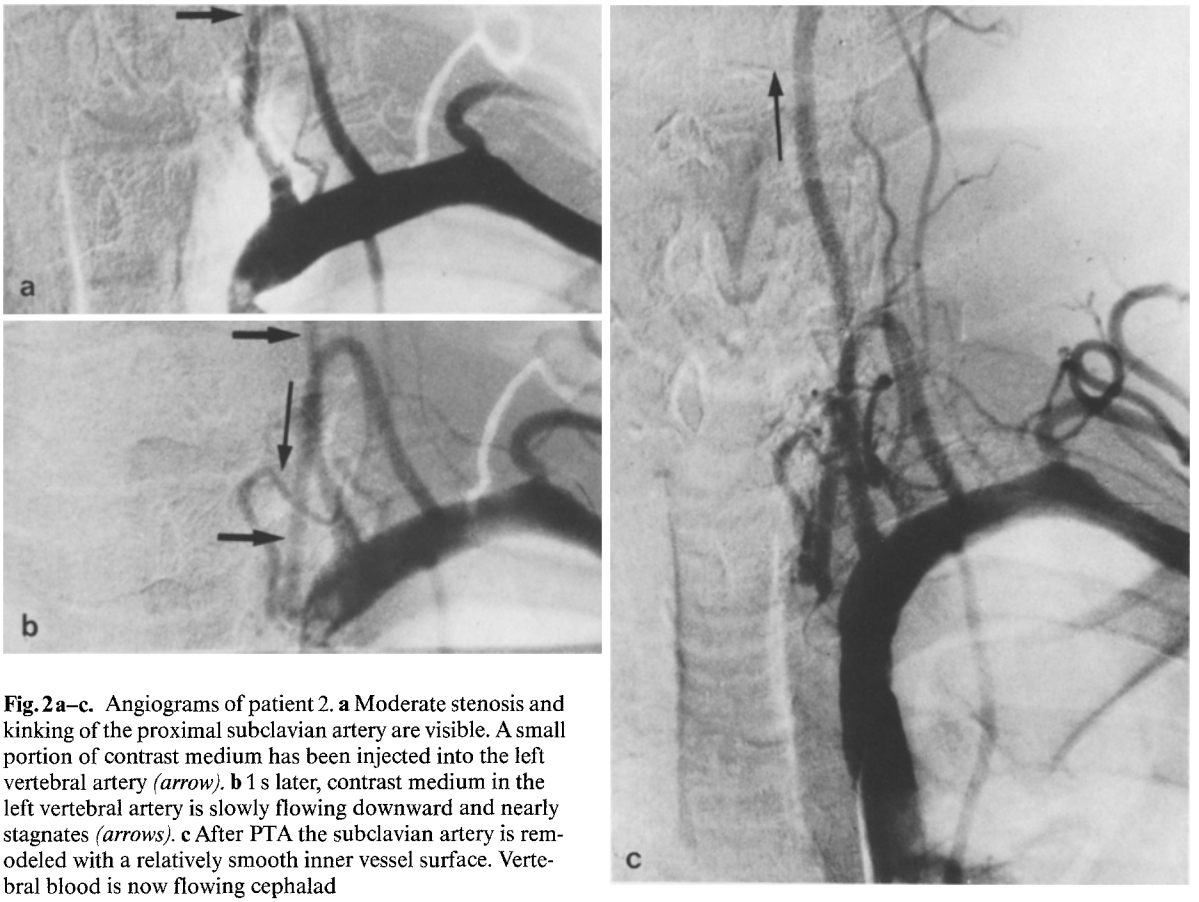

Fig. 2a–c. Angiograms of patient 2. **a** Moderate stenosis and kinking of the proximal subclavian artery are visible. A small portion of contrast medium has been injected into the left vertebral artery *(arrow)*. **b** 1 s later, contrast medium in the left vertebral artery is slowly flowing downward and nearly stagnates *(arrows)*. **c** After PTA the subclavian artery is remodeled with a relatively smooth inner vessel surface. Vertebral blood is now flowing cephalad

Performance of angiography and percutaneous transluminal angioplasty

During angiography, using the transfemoral route through a sheath set, the stenosis was negotiated with a straight guide wire. A 7-French balloon dilatation catheter (Olbert's type) [23] was advanced over the guide wire through the stenosis. The stenotic segment was dilated with several inflations of the 7 mm balloon. In both right-sided and left-sided stenoses the transfemoral route was chosen. The transaxillary approach was also attempted with two patients having subclavian artery occlusions.

Intra- and post-interventional vertebral blood flow monitoring

During the whole procedure, the ultrasound flow signals from the mastoidal slope of the homolateral vertebral artery were continuously recorded acoustically, as well as on a registration chart by one of the authors. A 4 MHz continuous wave Doppler device (D 800, Delalande) was used. The different alterations of subclavian blood flow and concomitant changes of vertebral flow profiles were related to the simultaneously performed balloon maneuvers (compare Figs. 6–8). Especially, the enforcement of the retrograde blood flow *during* PTA and re-establishment of antegrade vertebral flow *after* PTA were carefully registered. The temporal relation between these flow changes and the particular steps of the subclavian dilatation procedure were analyzed (compare details in Figs. 6–8).

Results

Twelve subjects suffering from latent or manifest subclavian steal syndromes due to moderate and tight stenoses or occlusions of the proximal subclavian arteries underwent PTA. Nine of them were successfully dilated without re-occlusion during a follow-up period from 1–7 months. In two patients with manifest subclavian steal syndromes due to proximal subclavian occlusions (stage IV) this method of intervention failed. The subclavian arteries could not be recanalized by either transfemoral or transaxillary approaches. In a third patient, the initial ultrasound diagnosis of highgrade proximal subclavian artery stenosis was proven by angiography to be correct (case 10). However, the patient turned out to have an additional occlusion of the distal part of the subcla-

Table 1. Sonographical, clinical and neuroradiological findings in patients with PTA for subclavian steal syndrome (SSS)

Case Age Sex (years)	Staging of SSS	Site of lesion	Concomitant vascular lesion	Complaints and clinical findings	PTA	Delay-period[a] (min)	Complications	Outcome
1 62 F	I[b] approx. 70% stenosis	left	no	vertigo, PRINDS[c] with left-sided hemiparesis; CT: no lesion	+	Primarily cephalad flow direction. Relative increase of cerebropetal flow after approx. 5	–	no further ischemic attacks
2 70 M	I[b] approx. 70% stenosis	left	carotid stenosis	vertigo, transient perioral and 5th finger dysaesthesia	+	indefinitive flow direction after PTA; cephalad blood flow immediately after retraction of catheter	–	no complaints
3 57 M	II approx. 80% stenosis	left	carotid stenosis	vertigo	+	approx. 1.5	–	no complaints
4 50 M	III approx. 90% stenosis	right	no	asymptomatic, but steadily increasing degree of stenosis during Doppler controls	+ transfemoral approach	approx. 5	transient paleness of one finger due to embolism	no complaints
5 63 F	III approx. 90% stenosis	left	no	attacks of vertigo, blurred vision, nystagmus	+	approx. 4	after symptom free period of 3 month duration again attacks of vertigo; gradually increasing stenosis at the site of PTA	no complaints after repeat PTA
6 68 M	III subtotal stenosis	left	no	asymptomatic, but evidence of brainstem lesion in orbicularis oculi reflex	+	approx. 4	–	no complaints
7 42 M	III subtotal stenosis	left		severe arm claudication, vertigo, headache, unknown hypertension	+	approx. 1.5	–	no complaints
8 70 M	III approx. 90% stenosis	right	slight carotid stenosis	severe periodic dizziness, drop attacks, recurring strokes with hind brain symptoms	+ transfemoral route	immediate reversal of vertebral flow after occurrence of subclavian occlusion	during PTA occlusion of subclavian artery distal to vertebral orifice	attacks less often
9 38 M	III subtotal stenosis; hypoplasia of stealing vertebral artery	left	no	headache, vertigo blurring of vision, arm claudication	+	more than 30 (not continuously documented)	–	completely free of symptoms
10 62 F	III approx. 90% stenosis; distal subclavian occlusion	right	no	vertigo arm claudication	+ transfemoral route; PTA attempted but balloon could not be placed	–	–	no improvement
11 63 M	IV	left	carotid stenosis	drop attacks, recurring dizziness	both transfemoral and transaxillary approach; penetration of occlusive lesion attempted but not achieved			no improvement
12	IV	left	internal carotid pseudoocclusion	dysarthria; progressive stroke due to hemispheric lesion	like case 10		death from myocardial infarction following reconstruction of internal carotid artery	

[a] Delay period = time between last balloon inflation and re-establishment of antegrade vertebral blood flow
[b] After detection of the "delay phenomenon" no other patients with stage I of SSS were treated with PTA
[c] PRIND = prolonged reversal ischemic neurological deficit

vian artery. This occlusion was well collateralized and therefore could not prevent the establishment of a vertebral steal mechanism. It prevented, however, an adequate placement of the balloon catheter and dilatation of the proximal stenosis.

Complications which may directly or indirectly be attributed to the dilatation procedure are listed in Table 1.

One patient experienced embolism as a complication of PTA of the proximal subclavian artery. He complained of transient pain and paleness of one finger of the homolateral hand lasting about 10 min. This symptom indicated an acute blockage of the arterial blood supply due to embolism into the finger arteries. However, no signs or symptoms of embolism into the vertebro-basilar system were found.

Representative examples of restoration of the vessel lumen by PTA are given in Figs. 2a–d and 3a–c. Angioplasty of left-sided moderate (case 2) and tight stenoses (case 6) are shown respectively. Fig. 4a–d gives an example of successful dilatation of a right-sided highgrade stenosis via the transfemoral route (case 4).

Continuous recording of the homolateral vertebral artery blood flow during PTA permitted the recognition of a remarkable phenomenon. This could

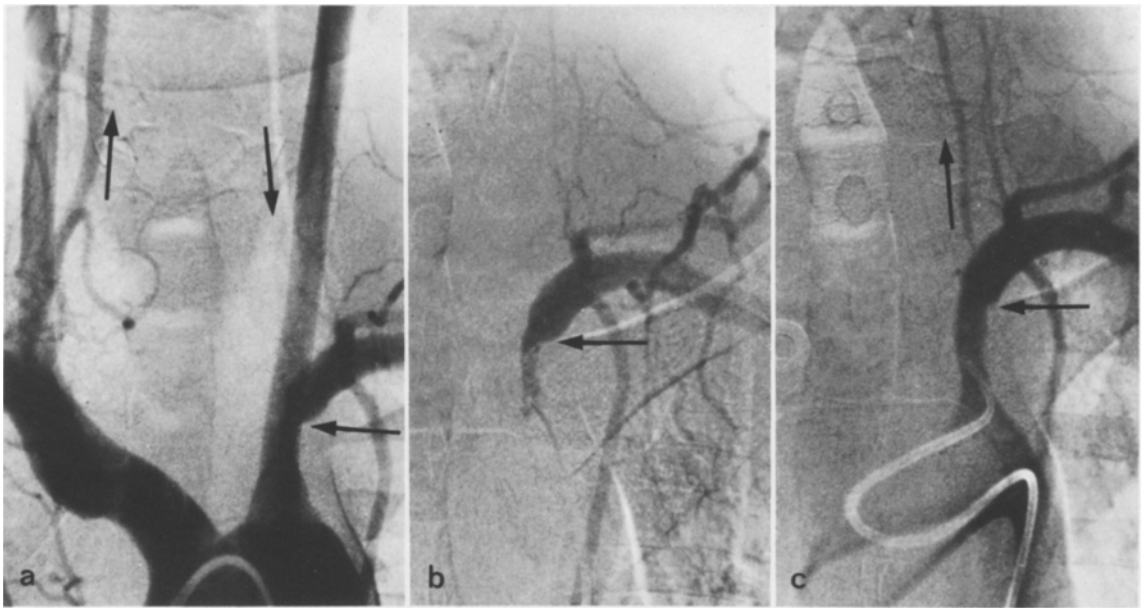

Fig. 3a–c. Angiograms of patient 9. **a** Subclavian steal via a hypoplastic left vertebral artery (light area) is visible. Horizontal *arrow* indicates the site of proximal subclavian stenosis. During arch aortography, the lesion seems to be of a moderate degree. **b** However, selective arteriogram shows the true severity of this highgrade stenosis *(horizontal arrow)*. **c** After dilatation with the help of a 7 mm balloon catheter, a slight plaque is still visible *(horizontal arrow)*. Vertebral blood flow is now antegrade *(vertical arrow)*; (6 weeks later, the flow had considerably increased and ultrasound findings were normal)

be observed more or less strikingly in most of the patients who underwent successful PTA. After application of the balloon dilatation, retrograde or to-and-fro blood flow within the stealing vertebral artery did not immediately change to an antegrade cephalad flow direction. This reversal, in fact, did not occur, although sufficient restoration of flow and blood pressure was found within the proximal subclavian artery. Only after a period of 20 s to more than 4 min, was antegrade flow re-established (Figs. 5 and 6). For the most part, however, the velocity profiles remained unaltered. The mean velocity of the blood flow was low during both systole and diastole when compared with the contralateral vertebral flow velocity. Immediate reversal was only seen in two patients. One suffered from a latent subclavian steal syndrome in the Ist stage (case 2), the other one was patient 8 (see Table 1). In this latter case, an accidental occlusion of the distal subclavian artery occurred during the dilatation maneuvre due to dissection of the non-stenosing plaque. Obviously, the abrupt blockage of the stealing territory permitted instant cephalad vertebral blood flow.

During the dilatation maneuver itself, the steal either became manifest or increased abruptly (Figs. 5 b, c and 6 e). This occurred at the moment when the balloon catheter was placed within the stenosing subclavian lesions.

In two patients, post-interventional angiography revealed marked increase of the diameter of the ipsilateral vertebral artery after cephalad blood flow had been re-established (Fig. 7 a, b).

Further Doppler ultrasound controls were performed repeatedly at intervals of 2 h, 2 days, 4 weeks and then every 3 months. Regularly, they revealed further improvement of vertebral blood flow on the side of the lesion. Flow velocity gradually increased up to normal values without further management.

Roughness of the intima at the site of angioplasty could be demonstrated during angiography immediately after PTA (compare Figs. 2 c, 3 c, 4 c). Abnormal turbulent flow within the subclavian artery could also be recorded by Doppler sonography. However, during post-interventional ultrasound reexaminations, remodeling of a smooth inner vessel surface became obvious as laminar blood flow re-appeared during Doppler sonographic studies.

No additional post-interventional short term or long term complications of PTA at the proximal subclavian segment occurred during the observation period in the 12 patients presented here.

Discussion

With the advent of percutaneous transluminal dilatation techniques [24] and improvement of balloon catheter technology [23, 25], application of PTA to nearly all vessel territories became more and more popular [1, 4, 5]. The fear of peripheral embolization

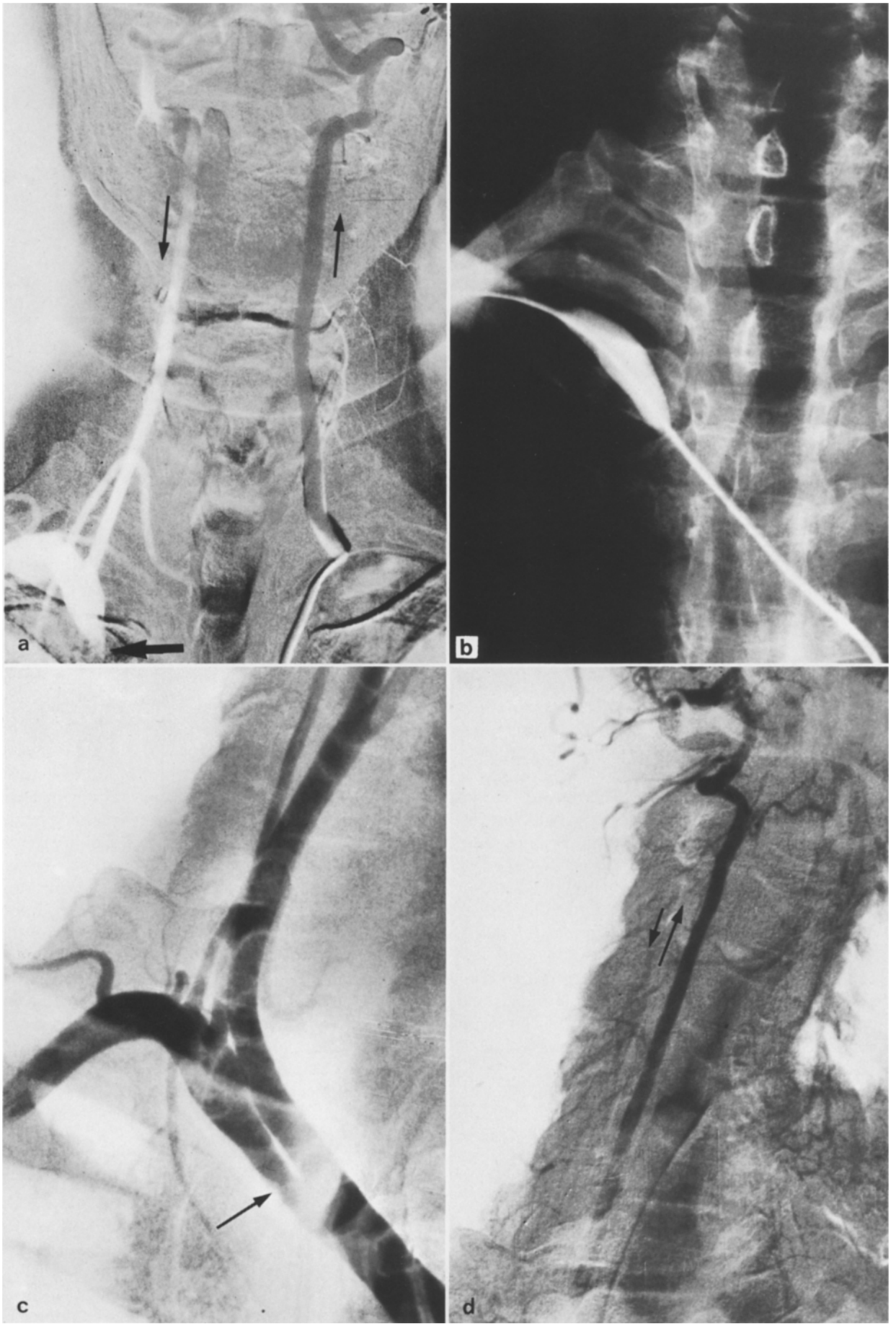

Fig. 4 a–d. Angiograms of patient 4. **a** Aortography (subtraction technique) reveals subclavian steal syndrome with blood flowing from the left vertebral artery (dark area) via the right vertebral artery (light area) to the right subclavian artery (*arrow* indicates the site of a highgrade stenosis of the proximal subclavian artery). **b** The inflated balloon catheter is placed within the stenotic segment. **c** Post-interventional control angiography shows moderate stenosis following PTA *(arrow)*. **d** The vertebral blood column slowly moves cephalad. (Fluoroscopy revealed to-and-fro movement) *(arrow)*

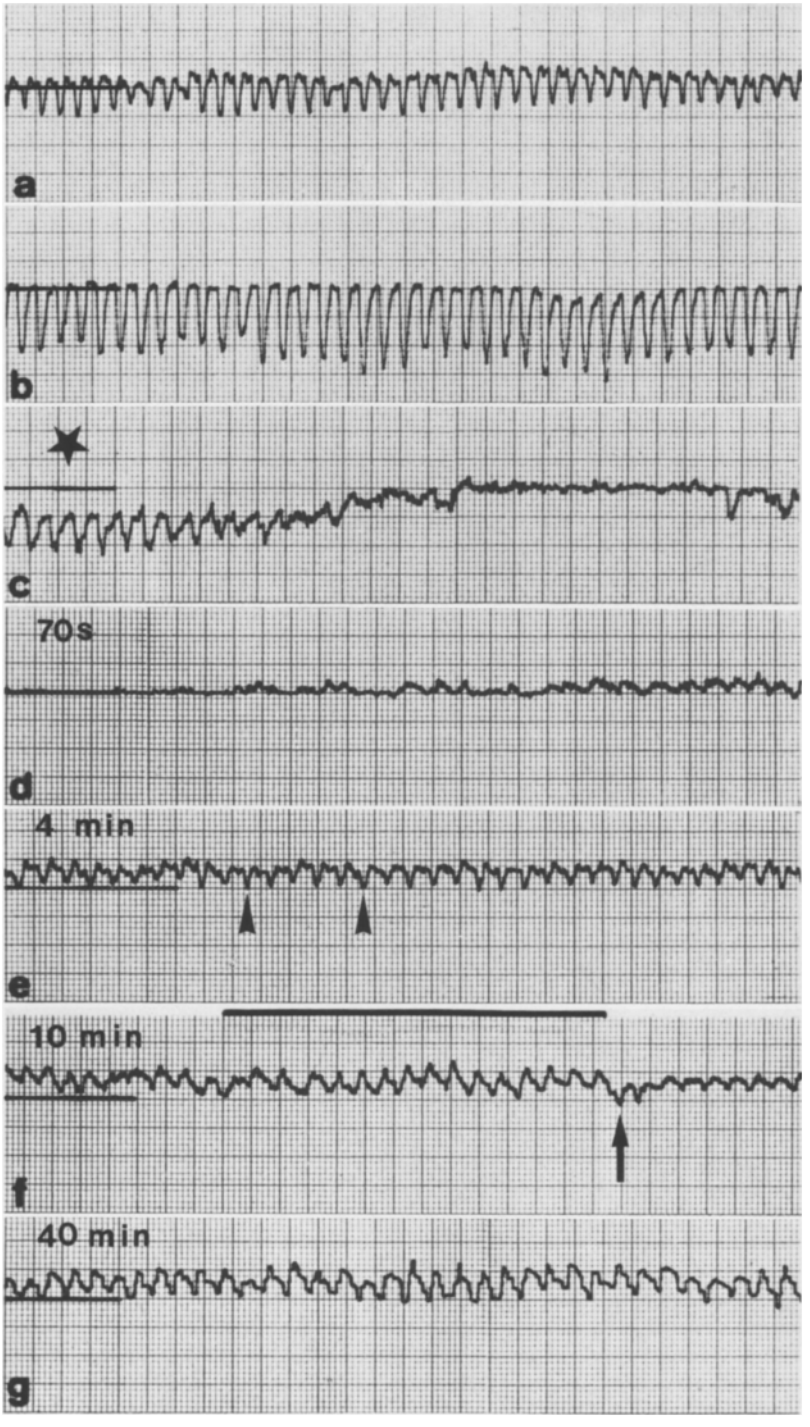

Fig. 5a–g. Vertebral blood flow before, during and after PTA for subclavian steal syndrome in case 7. **a** Latent subclavian steal (stage II) increases and becomes manifest **b** following insertion of the balloon catheter. **c** After a third balloon inflation *(asterisk)*, vertebral blood flow is only minimal. **d** Gradually, a cephalad blood flow is re-established after more than 70 s; **e** 4 min later, a slight pre-systolic deceleration of blood flow *(arrowheads)* is still visible; **f** 10 min later, blood flow is normal but can be influenced by compression of the brachial artery during measurement of blood pressure. Note short reversal of flow after deflation of the cuff *(arrow)*; **g** 40 min later cephalad blood flow has stabilized

prevented performance of PTA particularly at the supraaortic vessels. Embolic complications, however, were not as common a phenomenon as had been anticipated [26]. During PTA of a relatively large series of patients with proximal subclavian artery lesions, Mathias et al. did not produce any embolic complications within the hind brain circulation [19].

During Doppler monitoring of the vertebral blood flow, the ultrasound sonographic findings suggest that post-interventional delayed re-establishment of antegrade blood flow within the homolateral vertebral artery serves as a protective mechanism against vertebrobasilar embolism. Also in the case report of Bachmann et al. [17] reversal of vertebral blood flow occurred no sooner than "30–45 s" after restoration of the subclavian and brachial circulation (p 995) [18]. This was demonstrated by fluoroscopy. However, angiography does not seem to be the ideal

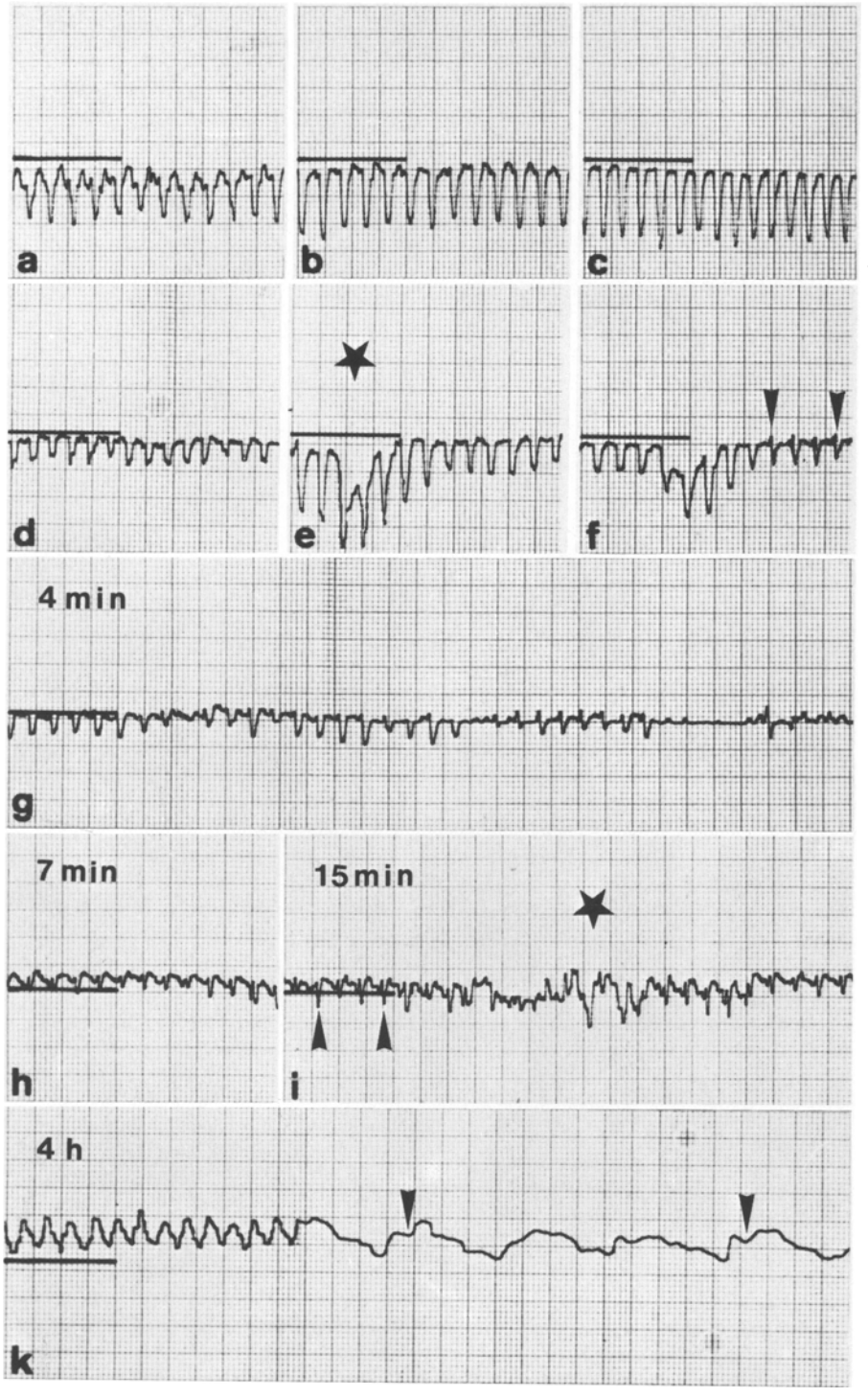

Fig. 6a–k. Vertebral blood flow before, during and after PTA for subclavian steal syndrome in case 6. **a** Diastolic flow velocity in manifest subclavian steal syndrome (stage III) is diminished following insertion of the guide wire **b** and furthermore with negotiation of the catheter **c**. After a first dilatation of the proximal subclavian stenosis the steal is already heavily reduced **d**. **e** After a second dilatation *(asterisk)* the balloon is deflated but not retracted into the aortic arch. **f** However, after a third dilatation the catheter is drawn into the aortic arch. The vertebral flow now begins to move cephalad *(arrowheads)*; **g** 4 min later, vertebral blood flow gradually shifts to cephalad flow direction; 7 min after the last balloon inflation **h** and 15 min later **i** the flow is still slightly perpendicular. Introduction of a normal French-7 catheter into the proximal subclavian artery accentuates the caudal flow compenent *(arrowheads)* (*asterisk* = passage of contrast medium); **k** 4 h later, flow profiles are nearly normal, but a slight systolic deceleration is still visible *(arrowheads)*

diagnostic tool. Due to a high injection pressure, the contrast medium might erroneously suggest cephalad flow direction despite a small but definite spontaneous downward flow (compare Fig. 2a and b). By contrast, Doppler sonography does not intervene in the unstable hemodynamic situation of the posterior circulation. Continuous insonation of the affected vertebral artery is a much more refined, highly sensitive and reliable method of analysing the intraarterial flow conditions. A net antegrade blood blow, i. e. a predominantly cephalad flow which is interrupted by a deceleration or downward movement of the

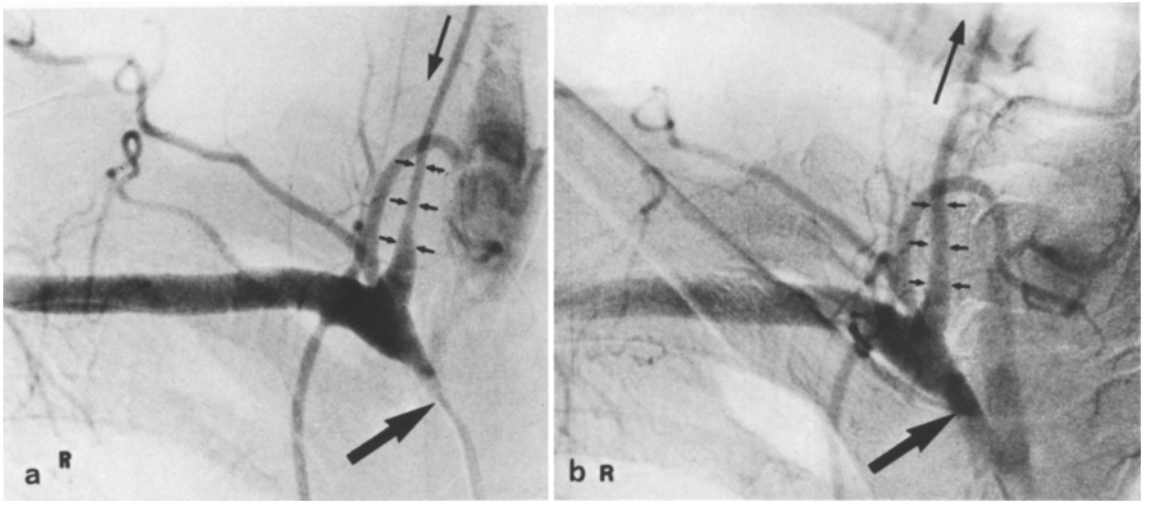

Fig. 7 a and b. Angiograms of patient 7. **a** Right-sided highgrade stenosis of the proximal subclavian artery *(arrow)* lead to subclavian steal with reversal of blood flow through a hypoplastic vertebral artery *(small arrows)*. **b** After PTA of the subclavian stenosis via the transfemoral route a slight narrowing of the subclavian artery is still visible at the site of the lesion *(arrow)*. Vertebral blood flow is now directed cephalad. Due to normal perfusion pressure the caliber of the vertebral artery has increased considerably *(small arrows)*

blood column during each cardiac cycle ("to-and-fro-movement" [21]) did not occur earlier than 20 s to several minutes after removal of the stenotic lesion. In several cases, complete antegrade vertebral blood flow was first seen the next day. Various factors (long duration and severity of the steal phenomenon, hypoplasia or stenosis of the stealing vertebral artery) seem to favor the delay of flow reversal.

Although peripheral emboli tend to occur immediately (within some seconds) after deflation of the balloon, one cannot completely exclude the later discharge of clot debris at a time when blood in the vertebral artery is already flowing in a cephalad direction. This occurred in one of our patients during the injection of the contrast medium which was performed for an angiographic study of the *post*-interventional state of the subclavian artery. A distal embolization of plaque debris could clearly be demonstrated by transient occlusion of the finger arteries.

The limbs would be relatively less vulnerable to embolic occlusions and ischemia because of their anastomotic network and their relatively low metabolism. The brain, however, does not tolerate embolism without functional disturbances or, in most cases, definite brain damage. This is especially true of the vertebrobasilar system. Larger emboli regularly cause a "top of the basilar"-syndrome [27] with severe visual, sensorimotor and mental deficits or may lead to mid-basilar occlusions. Smaller emboli often cause posterior lobe infarctions with visual field defects [28].

The pathophysiologic mechanism of the above described "delay phenomenon" remains unclear. However, there are some examples of a similar mechanism in other vascular territories where chronic collateral pathways have been established. Keller et al. [29] observed a shift toward normal perfusion of the ophthalmic artery only 24 h after endarterectomy of the occluded internal carotid artery had been performed. The suction effect of a stealing territory on the feeding vessels may be so strong, that even carotid-subclavian bypasses may fail to restore antegrade flow in the vertebral artery, but, in fact, may steal from the carotid artery [30].

It seems reasonable to suggest that PTA for subclavian steal syndrome should only be performed in stages II or III, i.e. when the vertebral artery definitely steals the blood downwards. Complete subclavian artery occlusion (stage IV) is ill-suited for PTA [5, 19]. Thus, stageing of the subclavian steal syndrome with the help of vertebral Doppler flow and excluding a subclavian artery occlusion are the neccessary prerequisites for the proper non-invasive selection of patients and reasonable timing of subclavian PTA.

Consideration of the delay phenomenon described above appears encouraging for the application of PTA in the treatment of subclavian steal syndrome. As conventional vascular surgery includes a considerable operative risk [31, 32] even if performed extrathoracically [33–36], the semi-invasive PTA seems to be a promising therapeutic alternative for patients with non-occlusive subclavian steal syndromes.

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