Recurrent laryngeal nerve paralysis associated with thoracic aortic aneurysm

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The association of vocal cord dysfunction with thoracic aortic aneurysm (TAA) has been noted in the cardiovascular and otolaryngologic literature.[1-6] A retrospective review of 168 cases of TAA was performed in order to: (1) define the natural history of associated recurrent laryngeal nerve paralysis (RLNP) and (2) propose mechanisms for the development of RLNP in operated and nonoperated aneurysms. Of 168 aneurysms, 5% manifested hoarseness secondary to RLNP. All had type I aneurysms. Only one patient regained vocal cord function after surgical treatment of the aneurysm. RLNP developed as a sequela of TAA repair in 12% of the patients managed surgically. RLNP associated with TAA type III repair had a higher incidence of recovery than paralysis that occurred after TAA type I repair (40% vs. 0% recovery). Sixty-six percent of all patients with permanently paralyzed larynges in this series attained glottic competence sufficient to avoid Teflon injection, and 27% of all RLNP associated with TAA in this series required Teflon injection for aspiration, severe dysphonia, or both. Seventeen percent of the patients with vocal cord paralysis associated with TAA recovered within 12 months. Aneurysm classification and pertinent anatomic relationships are discussed with reference to various mechanisms of recurrent laryngeal nerve paralysis. (OTOLARYNGOL HEAD NECK SURG 1990;102:140.)

Intrathoracic causes of vocal cord dysfunction have been described by Lewy,[2] Work,[3] Hirose,[4] and others. However, the incidence, pathophysiology, and clinical course of patients with recurrent laryngeal nerve paralysis (RLNP) associated with thoracic aortic aneurysm (TAA) have not been previously elucidated. In Lewy's series[2] of 144 cases of vocal cord paralyses treated by Teflon injection, only three were associated with TAA. Although Finkelmeier and Mentzer,[1] Stephens et al.,[5] and Urschel et al.[6] make reference to hoarseness associated with TAA in the cardiovascular literature, details of laryngoscopic examination and patient followup are lacking.

A retrospective review of 168 cases of TAA was performed in order to (1) define the natural history of associated RLNP and (2) propose mechanisms for the development of RLN in operated and nonoperated aneurysms.

METHODS AND MATERIALS

One hundred sixty-eight cases of TAA were evaluated at the Loyola University Medical Center from 1973 to 1987. A retrospective analysis was performed and the initial symptoms, aneurysm etiology, diagnosis, location, treatment, and presence of an associated vocal cord paralysis were documented.

Patients suspected of having TAA on clinical grounds underwent immediate chest x-ray or contrast aortography. Voice change, at presentation or after aneurysm repair, was evaluated by indirect laryngoscopy by a member of the otolaryngology department.

Medical management was the primary treatment for patients with chronic aneurysms, in patients older than 65 years of age with stable dilation, and in patients considered poor surgical candidates because of complicating medical problems. Therapy consisted of a combination of antihypertensive and diuretic agents, along with analgesic medication. Surgical repair was the primary treatment for patients with traumatic or acute dissections and patients with stable aneurysms considered medically fit for operation. The technique of repair varied with location and the extent of the aneurysm, but most procedures included proximal and distal aortic cross-clamping, resection of the dilated

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Table 1. Presenting manifestation of thoracic aortic aneurysm

	No.	%
Chest or back pain	100	60
Cardiac arrest or shock	20	12
Asymptomatic	16	10
Shortness of breath	12	7
Hoarseness	8	5
Other	9	5
Undocumented	3	1
	N = 168	100

Table 2. Aneurysm classification

Type	Location	No.	%
I	Ascending and arch	80	48
II	Ascending	7	4
III	Descending	70	42
Undocumented	—	11	6
		N = 168	100

vessel wall, and closure of the surgical defect by primary anastomosis or—more commonly—by dacron graft interposition.

Twelve aneurysms were documented at the time of autopsy. Seven of these deaths were caused by the aneurysm, whereas five were related to other causes.

RESULTS

One hundred twenty-eight of the 168 patients (77%) had symptoms referable to their aneurysm at the time of diagnosis. Chest or back pain was the most common initial symptom, occurring in 100 of the 168 patients (60%). Hoarseness or significant voice change was noted in eight patients (5%) (Table 1). Subsequent chest x-ray film and aortography documented the aneurysm in 93% of all cases.

Aneurysm classification was documented in 157 of the 168 cases. Types I and III aneurysms occurred in nearly equal frequencies, whereas type II aneurysms were very uncommon (Table 2). Of these 168 cases, 92 were treated by surgical resection of the aneurysm, 64 were managed with medical therapy, and 12 aneurysms were demonstrated at autopsy. Within the surgical group, postoperative left vocal cord dysfunction developed in 11 patients (12%), whereas eight patients initially manifested hoarseness due to vocal cord paralysis. Two of these eight patients were managed medically.

Table 3. Outcome in patients with RLNP as a presenting sign of TAA (N = 8)

Eventual outcome	Number
Contralateral VC compensation	4
Teflon injection	2
RLN recovery	1
Died	1
TOTAL	8

Table 4. Outcome in patients with RLNP following TAA repair (N = 11)

Eventual outcome	TAA type I	TAA type III
Contralateral VC compensation	3	3
Teflon injection	3	0
RLN recovery	0	2
TOTALS	6	5

All eight patients with dysphonia at initial presentation were of advanced age (greater than 60 years) and had type I aneurysms. One patient, who had presented with acute chest pain and a 1-day history of voice change, died. This patient died during arteriography from aortic arch rupture with pericardial tamponade. Of the remaining seven patients, four showed right vocal cord compensation at 1 year, one had resolution of a left vocal cord paralysis by 6 months, and two patients required Teflon injection for aspiration or hoarseness or both (Table 3).

In the 11 patients with postoperative dysphonia, hoarseness developed within the first postoperative week in each case. In this group, right vocal cord compensation was noted in six patients within 1 year of surgery, resolution of the left vocal cord paralysis occurred in two patients within 6 months, and three patients were injected with Teflon for persistent dysphonia. None of the paralyses that occurred after repair of six type I aneurysms recovered, whereas two of five paralyses that occurred after repair of type III aneurysm recovered within 12 months (Table 4).

RLNP did not develop in any patients with TAA type II in our series.

DISCUSSION

Aortic aneurysms are classified according to both location and period of onset. Whereas acute dissections are commonly seen with severe blunt trauma, chronic aneurysms are noted in cases of arteriosclerosis and aging. Thoracic aortic aneurysms, as seen in Fig. 1,

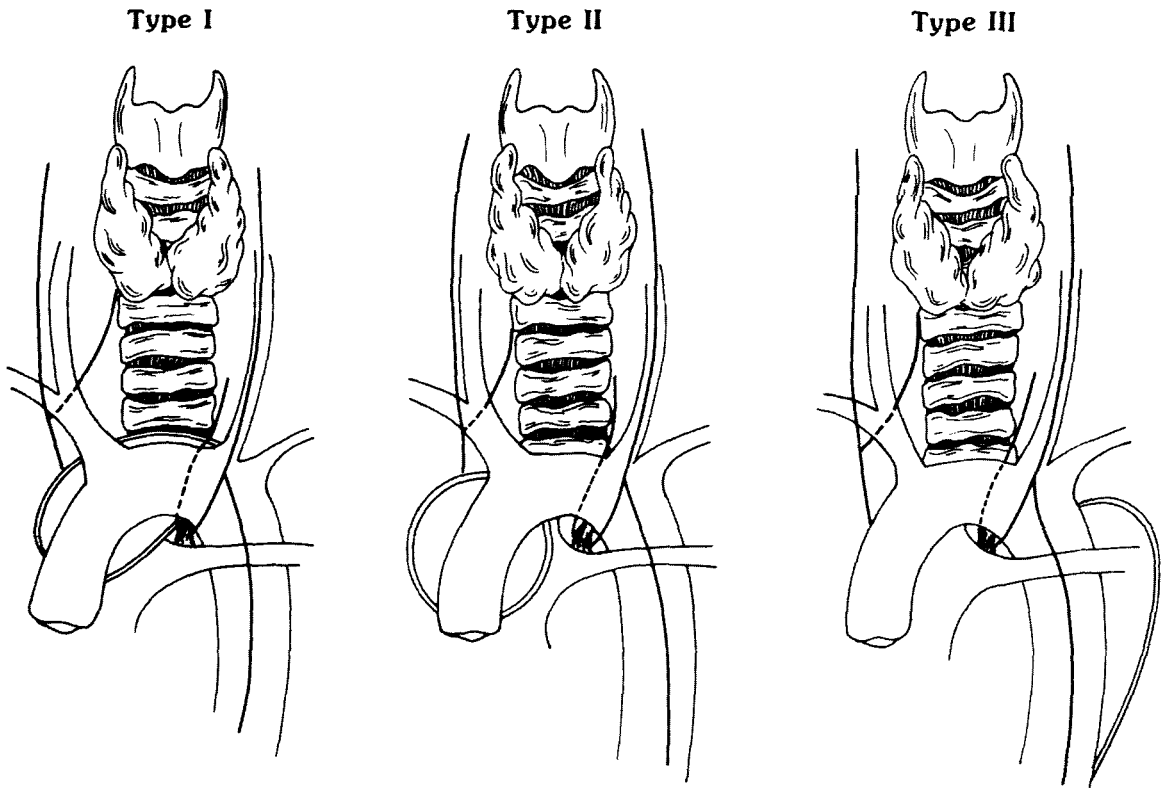

Fig. 1. Aneurysm type based on location and extent of involved aorta. Note the relation of the aneurysmal sac to the RLN.

Table 5. Possible mechanisms for recurrent laryngeal nerve trauma in cases of thoracic aortic aneurysms

Dysphonia a presenting *symptom*	Dysphonia as postoperative *complication*
Nerve compression by aneurysm	Nerve traction
Traction of nerve secondary to aneurysmal dilatation	Direct
Adherence of nerve after resolution of nonlethal bleeding	Secondary to sternal retraction
	Nerve trauma
	During aortic cross
	Laceration upon incision of aneurysm
	Incorporation of nerve in suture line

can involve the ascending root and arch (type I), be localized to the ascending segment only (type II), or involve the descending thoracic aorta only (type III). Types I and III are most common and occur in nearly equal frequencies.[7] In our series of 168 cases, the eight patients (5%) who manifested hoarseness all had type I aneurysms. Voice change developed postoperatively in 11 of 92 individuals (12%). These were equally divided between patients with types I and III aneurysms.

The anatomic relationship of the left RLN to the aortic arch and the ligamentum arteriosum renders the nerve vulnerable to compression and trauma by TAAs and their repair. The most obvious cause of hoarseness as an initial symptom with TAA is direct nerve compression by an expanding aneurysmal sac, with RLN traction resulting from further dilation. In addition, fibrosis in the vicinity of the RLN occurs after resolution of nonlethal aneurysm bleeding.[8] This perineural scarring can result in dysfunction of the vocal cord and hoarseness.

Postoperative dysphonia results from intraoperative trauma to the RLN, which is not always technically avoidable. Nerve traction can occur directly, or indirectly from sternal retraction, and can lead to various degrees of nerve damage. Difficult dissection in a scarred and fibrotic operative field may prevent identification of the RLN, and aortic cross-clamping,

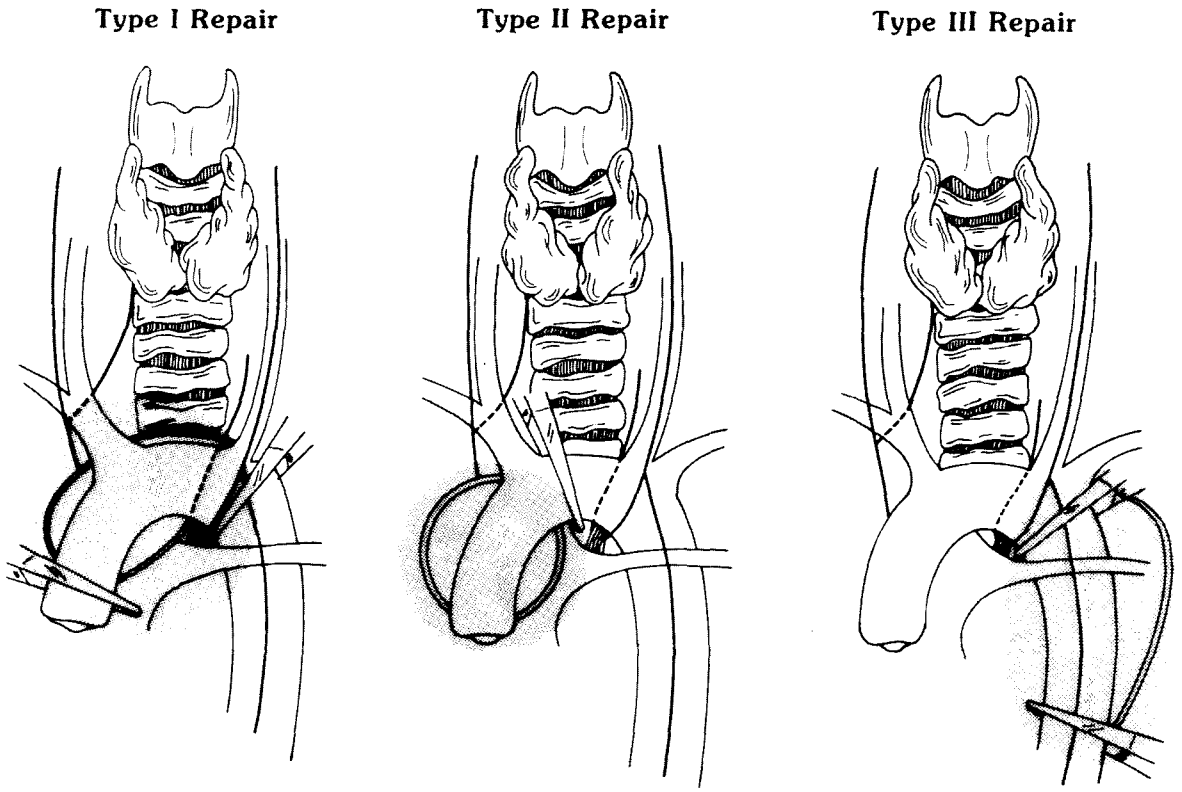

Fig. 2. Repair of TAA. Note the placement of aortic cross-clamps and the region of dissection in relation to the RLN.

laceration, inadvertent ligation, or incorporation of the nerve within the aneurysmorrhaphy suture line may result in neural trauma. Proposed mechanisms of nerve injury are outlined in Table 5.

The clinical course of vocal cord dysfunction associated with TAA probably depends upon the mechanism of nerve injury. Our data show no difference in recovery rate comparing preoperative and postoperative paralyses (12% vs. 18%), but significant difference in recovery rate comparing postoperative paralyses associated with type I and type III aneurysms (0% vs. 40%). Significance is confirmed using the *t*-test for independent variables and may be attributable to a difference in surgical technique required for repair of these aneurysms.

Repair of TAA type I requires dissection, cross-clamping, and aneurysmorrhaphy in the area of the ligamentum arteriosum (Fig. 2). The lack of recovery of vocal cord paralysis occurring in this group implies severe RLN damage.

Conversely, a 40% recovery rate was observed among paralyses that occurred during TAA type III repair, in which cross-clamping and dissection occurs distal to the ligamentum arteriosum (Fig. 2). Our data support the hypothesis that distal dissection and anastomosis of type III aneurysm repair make permanent RLN injury less likely than the proximal dissection and anastomosis used in type I aneurysm repair.

Glottic competence after permanent RLNP may be restored over the first 12 months by degrees of movement of the mobile cord across the midline and the passive medialization of the paralyzed cord attributed to atrophy, fibrosis, and contracture of the paralyzed vocalis muscle.[9] While the patient factors that predict the ability of the paralyzed larynx to regain glottic competence are not defined, our figures show 66% of permanently paralyzed larynges regain sufficient glottic competence to prevent aspiration (10 of 15).

The paralyzed cord of patients who did not acquire glottic competence were injected with Teflon paste. This group represents 27% of all paralyses associated with TAA in our series. The overall rate of RLNP recovery for all paralyses presenting preoperatively and postoperatively is 17% in our series.

SUMMARY

RLNP is associated with TAA: hoarseness may present as an initial symptom (5%) or as a postoperative complication (12%). Mechanisms of nerve trauma include aneurysmal compression or fibrosis along its course or intraoperative trauma to the nerve. The rate of resolution of RLNP is significantly higher after repair of TAA type III (40%) than type I (0%). The overall rate of RLNP recovery for all paralyses that were manifested preoperatively and postoperatively is 17% in our series.

Sixty-six percent of all patients acquired right vocal cord compensation at 1 year. Twenty-seven percent of all patients with RLNP associated with TAA in our series required Teflon injection for severe aspiration or dysphonia or both. An observation period of 12 months is advocated before Teflon injection is performed for dysphonia.

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