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Benign thyroid disease and vocal cord palsy

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The case notes of 2453 consecutive patients admitted for thyroid surgery and with successful preoperative laryngoscopy were examined retrospectively. Of the 2408 patients who had not had previous operations on the gland, 2321 proved to have benign pathology. A total of 29 patients had a preoperative vocal cord palsy of which 22 were associated with benign disease. Return of cord movement after surgery occurred in 89% of the patients with a benign goitre. We advocate routine preoperative laryngoscopy to detect vocal cord paresis. Such a finding with a goitre does not necessarily indicate malignancy. The recurrent laryngeal nerve should therefore be identified at surgery and preserved to allow for recovery of vocal cord movement.

Goitre with hoarseness is usually considered a portent of malignant thyroid disease with recurrent laryngeal nerve (RLN) involvement. However, cases of RLN palsy associated with benign thyroid pathology have been reported. Vocal cord palsy may also result from disruption of the vagus nerve and may be asymptomatic.

We therefore examined our case notes of patients admitted for thyroid surgery to ascertain how many had preoperative vocal cord palsies and how often this accompanied benign disease of the gland. The type of cord paresis was assessed in order to determine the likely mechanism of palsy. An abductor palsy suggests RLN involvement, leaving the action of the cricothyroid muscle unopposed and the cord in a paramedian position. An adductor palsy, with the cord in a lateral or cadaveric position, signifies loss of action of all the intrinsic laryngeal muscles including cricothyroid, thereby suggesting vagal or concurrent superior and recurrent laryngeal nerve involvement *(1)*.

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Materials and methods

The case records of 2453 consecutive patients undergoing thyroid surgery between 1947 and 1992, who had had successful preoperative laryngoscopy, were analysed retrospectively. Laryngoscopy had been performed indirectly using a mirror, or in the last 11 years with a flexible, fibreoptic nasendoscope when mirror examination had been unsuccessful. The patients all had their vocal cord assessments performed by a registrar, senior registrar or consultant. Before nasendoscopy became available 42 patients had failed indirect laryngoscopy and are therefore not included. They represent 2.4% of the prenasendoscopy total and are comparable with a 2.6% failure rate of routine vocal cord checks reported by Curley and Timms *(2)*.

The number of preoperative vocal cord palsies was sought. In all, 45 records were excluded, the patients having had previous thyroid surgery, leaving 2408 for study. The side of the palsy and its degree, whether partial or complete, was related to pathology and operative findings. The nature of the palsy, whether adductor or abductor, was also noted. Records were not available in all cases to ascertain whether the palsy was associated with dysphonia.

The group is highly selected, representing only those patients with disease requiring surgery. Postoperative, sequential laryngoscopy findings in those patients with benign disease were analysed for evidence of recovery of vocal cord movement. These patients were examined at 3 days after operation, monthly for 6 months and subsequently 6 monthly.

Table I. Comparison of the incidence of preoperative vocal cord palsy in benign and malignant thyroid disease, $n = 2408$

	Pathology	
Palsy	*Benign*	*Malignant*
Present	22 (0.95)	7 (8)
Absent	2299 (99)	80 (92)
Total	2321	87

Figures in parentheses are percentages

Results

In all, 22 patients with benign pathology had a vocal cord palsy. In only one case was bilateral involvement present. This patient had a cadaveric position of the right cord and decreased adduction of the left with no danger to the airway. Seven patients with thyroid malignancy had cord palsies. The relative incidence in each group is seen in Table I.

The relationship of benign pathology and operative findings to the side and degree of the palsy is illustrated in Table II. In two cases the recurrent laryngeal nerve was observed to be directly compressed by thyroid tissue, in a further three cases the nerve was seen to be displaced by a large nodule of a multinodular goitre, and in one case the RLN was clearly stretched by a cyst expanding into the mediastinum. Of the three cases with an adenoma, two were ipsilateral to the paralysed side. In 14 of the remaining 19 cases, the involved vocal cord was ipsilateral to the side of the more diseased lobe. Five of the palsies were complete and 17 were partial. Four patients (subjects 1, 5, 7 and 21) were found to have adductor palsies, two of which were complete, while the remaining subjects had abductor palsies.

Postoperatively, 19 patients in the benign group had completed records detailing sequential laryngoscopic findings. The nerve recovered and vocal cord movement returned in 17 patients (89%) (Table III). Six patients had regained cord movement by the 3rd postoperative day, five by between 4 and 7 months and seven by between 12 and 18 months. The two patients not regaining movement had been observed for 12 and 13 months, respectively.

Discussion

In standard surgical texts, goitre associated with a vocal cord palsy has classically been said to indicate thyroid gland malignancy *(3)*. In overall series of recurrent laryngeal nerve palsies, malignant disease is responsible for 17% to 38% of cases, one-half of these being caused by carcinoma of the lung *(4–9)*. The left RLN has a longer course and is therefore the side affected in approximately 75% of cases, particularly by carcinoma of the bronchus. Between 5% and 10% of malignancies responsible for RLN palsy are thyroid in origin *(7,9)*.

Non-surgical, blunt trauma is recognised as causing RLN palsy in 7% to 10% of patients *(8,9)*. Palsy may

Table II. Relationship of operative findings and pathology to the side and degree of vocal cord palsy in those patients with a benign goitre. Palsy is partial unless stated otherwise in parentheses

Subject	*Palsy*	*Pathology*	*Operative findings*	
1	L	MNG	Retrosternal	Large nodule compressing left RLN.
2	R	MNG	Retrosternal	Large posterior nodule on left side. Trachea grossly deviated to right.
3	R	MNG		Right lobe larger than left with deviated trachea to left.
4	L	MNG		Trachea deviated to right.
5	R(C)&L	MNG	Retrosternal	Trachea compressed.
6	R	MNG		Trachea deviated to right.
7	R	Adenoma		Right lobe tumour.
8	L	MNG		Left RLN displaced by a posterior nodule.
9	R(C)	MNG	Retrosternal	Right RLN displaced by a posterior nodule.
10	L	Adenoma		Right lobe tumour.
11	L(C)	MNG	Retrosternal	Gross enlargement to left lobe with a large cyst stretching left RLN in mediastinum.
12	L	MNG		Marked tracheal deviation to right due to large cystic mass on left.
13	L	Adenoma		Left lobe tumour.
14	R	MNG	Retrosternal	Tracheal deviation to left. Greater nodularity of right lobe.
15	R	MNG	Retrosternal	Large retrosternal extension of right lobe.
16	R	Hashimoto's	Retrosternal	Diffuse involvement.
17	L	MNG	Retrosternal	Displacement of left RLN and gross tracheal deviation by lower pole nodule.
18	R	MNG		Greater nodularity of right lobe.
19	R(C)	MNG	Retrosternal	Hard, right postermedial nodule compressing right RLN.
20	R	Graves'	Retrosternal	Trachea deviated to right.
21	L(C)	MNG		Trachea deviated to right.
22	R	MNG		Trachea deviated to right.

L = left, R = right, C = complete, MNG = multi-nodular goitre, RLN = recurrent laryngeal nerve

Table III. Preoperative vocal cord palsy and postoperative recovery of movement, $n = 19$

	Recovery	
Palsy	*Yes*	*No*
Complete ($n = 5$)	3	2
Partial ($n = 14$)	14	–

result from stretching of the nerve by an enlarged left side of the heart in congestive cardiac failure, by an aortic arch aneurysm or by a dilated pulmonary artery in mitral stenosis. Intubation may also be a cause due to pressure from an inflated endotracheal tube cuff *(10)*. Titche's series of 134 cases of RLN palsy contained only one associated with benign thyroid enlargement *(8)*. Willatt and Stell *(9)* referring to their 20-year series did not directly report a case related to benign thyroid disease but referred to three cases described by Worgan *et al. (11)*. However, two of these patients had had previous thyroid surgery.

Holl-Allen *(12)* reported a preoperative incidence of vocal cord palsy accompanying benign thyroid disease of 0.69% (eight out of 1156 patients). The nerve recovered after operation in three patients. Rueger *(13)* reviewed ten series of patients undergoing thyroid surgery for benign pathology from 1939 to 1969 and revealed an incidence of spontaneous, unilateral RLN paralysis of 0.7%. However, one of the included series had an incidence of paresis of 23%. If this is not included the overall occurrence falls to 0.63%. The author reported five further cases, two of which regained cord movement after operation.

Though in 1975 Pickleman *et al. (14)* suggested that vocal cord paralysis via RLN compression was a phenomenon increasingly recognised, few reports have been published in the subsequent English literature *(15–20)*. Four of these are isolated cases *(15–18)*. Three of the four regained cord movement after operation. McCall *et al. (19)* presented 14 out of 1500 patients (0.93%) with preoperative vocal cord paresis. However, these included patients with parathyroid pathology and thyroid malignancy. Only five of the palsies were solely associated with benign thyroid pathology, three of which recovered after operation. No proportion of the total number of patients with benign disease is given.

Of our 2321 patients with benign thyroid disease requiring operation, 22 (0.96%) had a preoperative vocal cord palsy. Given a recovery of movement in 17 out of the 19 (89%) patients with sufficient postoperative laryngoscopic follow-up, the palsies can accurately be attributed to benign thyroid pathology. This relationship is not as obvious in Holl-Allen's *(12)* and McCall *et al*'s *(19)* series in which only 38% and 60% of cases, respectively, recovered after operation. The two patients failing to regain movement were, unfortunately, lost to follow-up at 12 and 13 months after operation. It is possible they may have regained movement as the latest recovery noted in this series was at 18 months. However, improvement had already been noted by 1 year. Woodson and Miller *(21)* have highlighted the paucity of information on the course and prognosis of vocal cord palsy in previous series. They reported a further 103 patients, only four of whom recovered normal vocal cord function, all within 6 months. Of these patients, two had idiopathic palsies and two were the result of intubation. Four further patients regained partial movement, in three cases more than 2 years after onset. The cause of these palsies is not stated, however.

Mechanisms suggested for the production of vocal cord palsy include compression of the RLN and/or its blood supply between the goitre and cervical spine or trachea, stretching of the nerve, perinodular inflammation producing minute artery thrombosis and fibrosis involving the nerve, or direct involvement in a thyroiditis *(12,13)*. Gani and Morrison *(16)* suggested expansion of a cyst may involve the nerve in a pretracheal compartment syndrome. Release of tension at operation with restoration of nerve blood supply was seen as the reason for immediate postoperative return of cord movement at extubation. Quayle and Talbot *(18)* thought this was unlikely, suggesting that interruption of blood supply for more than 30 min would produce an ischaemic demyelination injury preventing immediate recovery. However, in our series restoration of movement was demonstrated at the first routine postoperative check at 3 days in six patients. Some of these patients may have regained movement immediately after operation. This suggests that palsy resulted from a neurapraxia, perhaps due to direct compression or stretching of the nerve, or may support reversible ischaemia as a cause.

Holl-Allen felt stretching of the RLN was only responsible in cases of retrosternal goitre *(12)*. In our series, four patients had an adductor palsy suggesting a lesion of the vagus nerve or superior and recurrent laryngeal nerves, with consequent paralysis of the cricothyroid muscle *(22)*. This may illustrate that stretching of the RLN does occur with a subsequent stretching of the vagus down from the base of the skull *(23)*. Only two of these patients had a retrosternal goitre. However, it may be difficult to assess correctly the true nature of a vocal cord palsy, particularly when partial, given that only 3–6 mm may separate the median, paramedian and cadaveric positions *(1)*. Consequently, clinical distinction between abductor and adductor palsies may be inaccurate.

It is recognised that unilateral RLN palsy associated with thyroid disease may be asymptomatic *(13,19)*. This may be so particularly with a slow growing goitre in which compensatory movement develops in the contralateral cord to that with a palsy. Work *(24)* reported that between 30% and 50% of RLN palsies in his series were asymptomatic, but no indication is given as to why these patients required a laryngeal examination and this percentage is elevated above more recent experience. Curley and Timms *(2)* had no cases of clinically unsuspected vocal cord palsies in their preoperative checks.

However, McCall *et al. (19)* had four out of 14 palsies associated with thyroid or parathyroid pathology that were asymptomatic.

We strongly advise routine, preoperative laryngoscopy of all patients undergoing thyroid surgery, to confirm and document the side(s) and nature of clinically suspected palsies and to detect the rare asymptomatic palsy. In our series paralysis was present in 1.2% of patients admitted for thyroid surgery and, furthermore, in 0.96% of those found to have benign pathology. Preoperative vocal cord palsy should not be assumed to indicate malignancy. The RLN should therefore be identified in these cases and preserved at operation. In 89% of our benign cases, vocal cord movement recovered after operation.

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