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Diaphragm strength in patients with recent hemidiaphragm paralysis

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ABSTRACT Eleven patients with unilateral diaphragm paralysis of recent onset were studied to investigate the effect of the paralysis on inspiratory muscle function. Nine of the patients had noticed a decrease in exercise tolerance, which was not explained by any other pathological condition. Hemidiaphragm dysfunction was confirmed by the demonstration of a greatly reduced or absent transdiaphragmatic pressure on stimulation of the phrenic nerve in the neck, by means of surface bipolar electrodes (unilateral twitch Pdi), compared with normal values on the contralateral side. Transdiaphragmatic pressure was 44·6% (9·4%) predicted during a maximal sniff and 30·3% (16·8%) predicted during a maximal static inspiration against a closed airway, confirming diaphragm weakness. Maximum static inspiratory mouth pressures were also low (61·7% (12·7%) predicted), consistent with a reduction in inspiratory muscle capacity. Phrenic nerve conduction time was prolonged on the affected side in nine patients, consistent with phrenic nerve dysfunction, whereas on the unaffected side it was normal. It is concluded that recent hemidiaphragm paralysis causes a reduction in transdiaphragmatic pressure that is associated with a reduction in maximum inspiratory mouth pressure. Phrenic nerve stimulation is a useful technique with which to confirm and quantify hemidiaphragm dysfunction. Measurement of phrenic nerve conduction time provides useful information about the underlying pathology.

Pulmonary function has been extensively studied in patients with unilateral diaphragmatic paralysis,[1] although the effect on inspiratory muscle function has been little reported. One recent study investigated patients with longstanding unilateral diaphragm elevation[2] and found variable effects. Maximal transdiaphragmatic pressure (Pdimax) was normal in 27% and maximal inspiratory mouth pressure (Pimax) normal in 40% of patients.[2] It was suggested that the normal respiratory muscle strength found in some patients was due to mechanisms such as hypertrophy of the contralateral hemidiaphragm and increased stiffness of the paralysed side, which compensated for the unilateral diaphragm paralysis. In this study, however, hemidiaphragm paralysis was not necessarily present in all cases, as it was inferred from the presence of an elevated hemidiaphragm on the chest radiograph and a positive result in the sniff test on fluoroscopy, both of which are recognised to be associated with a considerable false positive rate.[3]

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We have therefore studied maximal respiratory pressures and transdiaphragmatic pressures in 11 patients in whom hemidiaphragm elevation was known to be recent, to investigate its effect on diaphragmatic and inspiratory muscle function before any compensatory mechanisms were likely to have occurred. We confirmed hemidiaphragm paralysis or severe dysfunction by measuring transdiaphragmatic pressure during stimulation of the phrenic nerve in the neck (unilateral twitch Pdi). In addition, we assessed phrenic nerve function by measuring phrenic nerve conduction time.

Methods

We studied patients with a raised hemidiaphragm of recent onset (table 1). All had had a normal chest radiograph within the previous year, confirming that paralysis was recent and excluding congenital eventration. Patients 1–4 had developed unilateral diaphragm paralysis after cardiac surgery, presumably as a result of phrenic nerve injury; patients 5–9 had developed it after a chest infection, and patient 10 after manipulation of his neck. In patient 11 the cause was unclear; it

Table 1 *Details of patients with raised hemidiaphragm*

No	*Side*	*Cause*	*Dyspnoea MRC scale*	*Orthopnoea*	*Other pathological conditions*	*FEV_1/FVC (%)*
1	L	Aortic VR	4	0	Emphysema	55
2	L	Aortic VR	2	0	Ex-smoker	78
3	L	Aortic VR	4	+	Emphysema	38
4	R	CABG	4	+ +	Ischaemic heart disease	67
5	L	Infection	3	0	Moderate asthma	59
6	L	Infection	2	0	Bronchial hyperreactivity	75
7	R	Infection	1	0	None	90
8	L	Infection	2	0	None	78
9	L	Infection	2	0	Ex-smoker	73
10	L	Cervical	1 (singing)	0	None	81
11	R	Idiopathic	3	0	Chronic bronchitis	51

VR—valve replacement; CABG—coronary artery bypass graft.

had started at some time within 12 months of a normal chest radiograph.

In three of the 11 patients no other pathological condition was present and in two (Nos 2 and 9) there were mild spirometric abnormalities assumed to be related to smoking. One patient (No 5) had moderately severe asthma and another (No 6) had bronchial hyperreactivity after the chest infection during which the unilateral diaphragm paralysis had developed. Two patients had emphysema (No 1 and 3), and one moderate airways obstruction related to chronic bronchitis (No 11). One patient (No 4) has ischaemic heart disease.

Four patients underwent fibreoptic bronchoscopy to exclude bronchogenic neoplasm.

Breathlessness was expressed in terms of the Medical Research Council (MRC) disability scale of 1 (no breathlessness) to 5 (breathlessness on minimal exertion).

Fluoroscopic diaphragm screening was carried out in all patients, supine, during a maximal sniff. Vital capacity (VC) was measured in both the sitting and the supine posture with a rolling seal spirometer (Spiroflow), the change in VC when the patient became supine being expressed as a percentage of the value for the sitting posture ("supine fall in VC"). Absolute lung volumes were measured in a constant volume whole body plethysmograph. The forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) were measured from a forced expiration into the rolling seal spirometer. All volumes were corrected to BTPS. The transfer factor for carbon monoxide (TLCO) and transfer coefficient (KCO) were determined by the single breath method.

Global respiratory muscle strength was assessed by measuring static maximal expiratory (PEmax) and inspiratory (PImax) pressures at the mouth. Efforts were initiated at total lung capacity and residual volume respectively, the method being based on that of Black and Hyatt,[4] and the results were compared with the values for 80 normal subjects studied in this laboratory. Measurements were repeated until a reproducible value was obtained for each (usually after about six attempts). Oesophageal (Poes) and gastric (Pgas) pressures were measured with balloon catheter systems connected to Validyne pressure transducers (model MP 45–1; range ± 150 cm H_2O). Each balloon was 10 cm long and 3·5 cm in circumference. The oesophageal balloon was positioned in the mid oesophagus and contained 0·5 ml of air. The tip of the gastric balloon was positioned 65 cm from the nares and the balloon contained 2·0 ml of air.

Transdiaphragmatic pressure (Pdi) was derived electronically according to the equation Pdi = Pga-Poes[5] but with Pdi at resting end expiration as zero reference point. Diaphragm strength was assessed by measuring Pdi during a maximal static inspiratory effort at residual volume against a closed valve (PdiPImax) and during a short, sharp maximal sniff (sniff Pdi).[6] Sniffs were performed at functional residual capacity without a noseclip and were repeated with pauses of at least 10 seconds between each sniff until a reproducible value of peak sniff Pdi was obtained (usually about six sniffs). Ten consecutive sniffs were then recorded and the greatest value was taken as the maximal sniff Pdi.

The phrenic nerves were stimulated in the neck with bipolar surface electrodes at the posterior border of the sternomastoid muscle by means of square wave impulses 0·1 ms in duration, with a frequency of 1 Hz and supramaximal voltage. Each phrenic nerve was stimulated in turn and the twitch Pdi measured for each side. Bilateral twitch Pdi was then measured during stimulation of both phrenic nerves simultaneously. On average 30 maximum twitches were measured on each occasion, the actual recorded result being the mean of the 10 largest twitches. Phrenic nerve conduction time (inversely related to nerve velocity) was measured with surface electrodes in the 7th and 8th intercostal spaces to record diaphragm evoked action potentials from the electromyogram and taken as the time interval between the initial

stimulation and the beginning of the action potential on the electromyogram.

Signals were recorded on an eight channel strip chart recorder (Mingograph 800, Siemens) and stored on a magnetic tape recorder (Racal Store 7) for later playback and analysis.

Results

Breathlessness expressed according to the MRC scale is shown in table 1. Only two patients had noticed no breathlessness or decrease in exercise tolerance (Nos 7 and 10) and one of these did notice slight breathlessness while singing. The other nine patients had all noticed an increase in breathlessness that was not explained by a deterioration in other pathological conditions.

Under fluoroscopic screening when supine all patients showed paradoxical movement of the affected hemidiaphragm on sniffing. In seven patients vital capacity was less than 75% predicted (table 2), but in the four remaining patients it was normal.[7] The mean fall in vital capacity with the change to the supine posture was within normal limits in all patients (12% (8%), normal <25%[8]). FEV_1 ranged from 35% to 101% (mean 65% predicted) and FEV_1/FVC was less than 75% in six patients (table 1). Total lung capacity was measured in nine patients (table 2) and was reduced to less than 75% predicted[7] in two (Nos 4 and 6). Residual volume was greater than 125% predicted[7] in two patients (Nos 3 and 5) and in each this was attributable to airways obstruction. TLCO was less than 75% predicted[7] in six patients out of nine; whereas KCO, also measured in nine patients, was reduced only in the two patients with emphysema (Nos 1 and 3).

Mean PEmax was 112 cm H_2O (95% pred), indicating normal expiratory muscle strength (table 3). Only one patient, who had had a thoracotomy (No 4), had a

Table 2 *Lung function in the 11 patients*

Patient No	*Vital capacity (% pred)*	*Supine fall in VC (%)*	*TLC (% pred)*	*RV (% pred)*	*TLCO (% pred)*	*KCO (% pred)*
1	2·9 (91)	10	94	117	39	59
2	2·6 (68)	0	*	*	88	*
3	2·5 (72)	22	87	126	51	59
4	1·7 (36)	*	56	90	53	132
5	2·5 (88)	10	114	145	108	103
6	4·2 (69)	22	74	78	70	108
7	4·6 (135)	17	*	*	*	*
8	2·6 (68)	19	93	121	82	119
9	1·9 (61)	0	85	107	71	91
10	3·7 (73)	8	104	101	113	133
11	2·9 (75)	10	82	78	56	88
Mean	2·9 (76)	11·8	87·7	107·0	73·1	99·1
SD	0·9 (24)	8·1	16·8	22·7	24·7	27·7

*Data not available.

Table 3 *Respiratory muscle strength: results of voluntary manoeuvres in the 11 patients*

		PEmax		*Pimax*		*Sniff Pdi*		*PdiPimax*	
Patient No	*Sex*	*cm H_2O*	*% pred*	*cm H_2O*	*% pred*	*cm H_2O*	*% pred*	*cm H_2O*	*% pred*
1	M	84	70	52	60	67	45	35	32
2	M	88	70	56	65	65	44	22	20
3	F	92	96	45	66	50	41	40	62
4	M	77	57	27	31	35	24	5	5
5	F	64	71	52	75	57	47	22	34
6	M	104	112	52	72	85	57	7	6
7	M	188	147	64	77	50	34	40	37
8	M	236	182	48	57	82	55	50	46
9	F	80	83	42	63	50	41	22	34
10	M	124	85	52	61	87	59	22	20
11	M	95	69	45	52	58	39	40	37
Mean		112·0	94·7	48·7	61·7	62·3	44·6	27·7	30·3
SD		52·9	38·4	9·4	12·7	16·7	9·4	14·4	16·8
Normal values*: mean SD									
	M	154	(82)	114	(36)	148	(24)	108	(30)
	F	94	(33)	71	(27)	121	(25)	65	(31)

PEmax, Pimax—static maximum expiratory and inspiratory pressure measured at the mouth; sniff Pdi—transdiaphragmatic pressure measured during a short, sharp maximal sniff; PdiPimax—transdiaphragmatic pressure measured during a maximal static inspiratory effort at residual volume against a closed valve.
*From Leech *et al*[13] and Miller *et al*.[6]

Table 4 *Phrenic nerve stimulation: results in the 11 patients*

Patient no	*Affected side*	*Unilateral diaphragm pressure (cm H_2O)*		*Bilateral diaphragm (cm H_2O)*	*Conduction time (ms)*	
		Affected side	*Normal side*		*Affected side*	*Normal side*
1	L	0	6	6	12	8
2	L	3	9	10	7	7
3	L	2	6	6	16	7
4	R	0	8	9	†	8
5	L	5	7	13	18	7
6	L	2	17	17	17	9
7	R	0	15	17	15	6
8	L	2	8	11	8	7
9	L	0	12	12	12	8
10	L	0	6	6	35	10
11	R	0	7	9	11	8
Mean	1·3	9·3	11·1			
SD	1·7	4·0	3·5			
Normal values (range)*		*Diaphragm pressure* R5·4–12·4 L7·8–15·3 Bilateral 14·6–33·4		*Conduction times* 6·0–9·5 5·5–9·5		

*From Mier *et al.*[9 10]
†M wave too small for determination of conduction time.

PEmax value significantly less than predicted. Mean maximum inspiratory muscle strength (PImax), however, was only 49 cm H_2O (62% pred), consistent with inspiratory muscle weakness. Diaphragm strength, as measured by sniff Pdi (mean 62 cm H_2O, 45% pred) and PdiPImax (mean 28 cm H_2O, 45% pred) and PdiPImax (mean 28 cm H_2O, 30% pred), was also reduced in all 11 patients.

Unilateral twitch Pdi was reduced or undetectable on the affected side, confirming hemidiaphragm dysfunction in all 11 patients (table 4), whereas on the unaffected side it was normal. Mean bilateral twitch Pdi was also reduced, confirming that overall diaphragm function was impaired. Phrenic nerve conduction time was prolonged on the affected side in nine out of 11 patients (table 4), consistent with phrenic nerve dysfunction. It was normal in two: patient 2, studied one year after aortic valve replacement, and patient 8, who had had a chest infection. It was normal on the unaffected side in all 11 patients.

Discussion

This study confirmed the presence of appreciable inspiratory muscle weakness due to diaphragm dysfunction in 11 patients with a raised hemidiaphragm of recent onset. Unilateral hemidiaphragm dysfunction was confirmed by the greatly reduced twitch pressure in response to phrenic nerve stimulation on the affected side. This appears to be a useful test with which to confirm and quantify hemidiaphragm dysfunction or paralysis and is more discriminating than fluoroscopy, which cannot quantify the degree of weakness and has a false positive rate of 6% in normal subjects.[3] We were unable to locate the phrenic nerves in a twelfth patient, a woman with considerable obesity, who was therefore excluded from the analysis. This was not regarded as a false negative result since it was clear from our inability to obtain a diaphragmatic electromyogram on either side that the phrenic nerves had not been located.

Our study showed inspiratory and diaphragmatic weakness in all patients confirmed to have hemidiaphragm paralysis or dysfunction of recent onset. Impaired muscle function was more consistently present than in the recent study by Lisboa *et al* of patients with longstanding hemidiaphragm elevation.[2] This discrepancy is, however, compatible with their suggestion that the apparently normal inspiratory function found in some patients with longstanding paralysis may be due to hypertrophy of the contralateral hemidiaphragm as this would not have had time to occur in our patients.

It is sometimes thought that unilateral hemidiaphragm paralysis does not give rise to functional abnormality or symptoms. Breathlessness has, however, been reported in 10–24% of such patients.[11] In the study of Lisboa *et al*[2] all five patients with diaphragm weakness due to longstanding hemidiaphragm paralysis resulting from phrenic nerve crush complained of dyspnoea, although extensive tuberculous sequelae may also have contributed. Of their 10 patients in whom the cause of the longstanding hemidiaphragm elevation was unknown, both the patients with associated cardiopulmonary disease and three of the eight patients with no associated path-

ological condition were breathless.

In some of our 11 patients breathlessness may have been the result of inspiratory muscle dysfunction superimposed on another pathological condition, such as airways obstruction, which increases the demands of the respiratory muscles. All patients had, however, noticed a change in breathlessness after the onset of the unilateral diaphragm paralysis that was not related to a deterioration in the lung disease.

Patients may be more aware of breathlessness at the time of onset of unilateral diaphragm paralysis; for instance, patient 7 was initially breathless but had improved by the time of study three months later, even though he still had residual inspiratory muscle weakness.

Pimax may already be reduced in patients with severe chronic airways obstruction[12] owing to the mechanical disadvantage of increased lung volume. This was unlikely, however, to be contributing significantly to inspiratory muscle weakness in our patients since total lung capacity was not increased by more than 14% of the predicted value.[12]

In conclusion, measurement of unilateral twitch Pdi during phrenic nerve stimulation is a useful test with which to confirm and quantify unilateral hemidiaphragm dysfunction. Recent onset of hemidiaphragm paralysis or dysfunction is associated with a reduction in maximal transdiaphragmatic pressure, leading to reduced inspiratory capacity. This may explain the increase in breathlessness and decrease in exercise tolerance associated with the condition, which are exacerbated by any additional pathological condition, thus increasing the demands of the respiratory muscles. Prolongation of phrenic nerve conduction time confirms a neuropathic cause.

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