immigration. These factors have been described previously by other authors as predictors of compliance failure.[12,13]

In conclusion, TLTI compliance in our center was satisfactory. Although the appearance of AEs was very common, these were easily resolved with close monitoring by expert personnel and easy access to the clinic, facilitating completion of the TLTI.

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Cardiorespiratory Side Effects in the Clipping Technique for the Treatment of Axillary and Palmar Hyperhidrosis☆

Efectos secundarios cardiorrespiratorios en la técnica del pinzamiento para el tratamiento de la hiperhidrosis palmar y axilar

To the Editor:

The surgical treatment of primary palmar and axillary hyperhidrosis (HH) consists of interruption of the thoracic sympathetic nerve. The most common intervention is sympathetic chain lysis or sympathectomy.[1,2] The clipping technique was designed to allow reversal of the intervention in the case of severe adverse effects, primarily compensatory HH.[3] The main aim of this study was to evaluate the effects of this technique on cardiopulmonary function.

This was a prospective study, approved by the Clinical Research Ethics Committee of Hospital Universitario de Gran Canaria Dr. Negrín Centro. All patients signed an informed consent form before participation. The study variables were respiratory and cardiovascular side effects. Patients aged between the ages of 14 and 40 years, with palmar or palmoaxillary HH and no history of smoking or cardiorespiratory disease were included.

Study patients were treated with a surgical technique of clipping at the T3 (palmar HH) or T3–T4 (palmoaxillary HH) level. Cardiopulmonary function was studied before and 6 months after the intervention. The following tests were performed: forced spirometry, measurement of lung volumes and airway resistance, calculation of CO diffusion, and a maximum incremental cardiorespiratory exercise test and stress test using a cycle ergometer.

Quantitative variables were analyzed using the t test for paired data and repeated measurements using analysis of variance. Categorical variables were compared with the Chi-square or Fisher's exact test. The SPSS 15.0 statistical package (SPSS Inc., Chicago, IL, USA) was used. Differences with a P value <.05 were considered statistically significant.

We analyzed 31 patients with an average age of 21.81±4.87 years who underwent surgery between 2013 and 2015. There were no postoperative complications. Six months after surgery, a significant decrease was observed in $FEF_{25\%–75\%}$ (−5.6%), and no differences were found in FVC, FEV_1, lung volumes and airway resistance. CO diffusion decreased significantly (−6.4%). The stress test showed a significant decrease in maximum minute ventilation (−12.2%), and in heart rate at peak effort (−3.9%) and at 2 min during recovery (−6.2%). No significant differences were found in oxygen consumption. Systolic and diastolic arterial pressures were reduced at peak effort (−11.5% and −7.1%, respectively), as was diastolic blood pressure at rest (−8.1%). All patients completed the pre- and post-clipping exercise tests with no significant symptoms. These data are summarized in Table 1.

Similarly to other sympathectomy techniques,[4–6] this study showed that clipping the sympathetic chain at the T3 and T3–T4 level causes significant changes in cardiopulmonary function. We found a decrease in $FEF_{25\%–75\%}$, probably reflecting an increase in bronchomotor tone due to the altered balance between sympathetic and parasympathetic innervations. However, in previous studies,[4,5] we found a decrease in FEV_1 and $FEF_{25\%–75\%}$, in line with the results of other authors,[7] showing that changes in spirometry are less significant with clipping than with conventional sympathectomy. We did not note any changes in lung volumes, although the non-significant increase in airway resistance would support the hypothesis of an increase in bronchomotor tone. The decline

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Table 1
Relevant Findings in Cardiopulmonary Function at Baseline and 6 Months After the Intervention.

Variable Cardiorespiratory Function	Baseline (Mean±SD)	At 6 Months (Mean±SD)	*P*
FVC (L)	4.1±0.7	4.1±0.8	ns
FEV_1 (L)	3.6±0.6	3.6±0.7	ns
$FEF_{25\%–75\%}$ (% pred)	94.1±21.1	88.5±19.3	.01
DLCO (mL/min/kPa)	8.4±1.8	7.7±1.9	.004
DLCO (% pred)	86.3±13.6	79.9±13.2	.01
HR_{rest} (bpm)	84.7±14.3	81.01±14.6	ns
HR_{max} (bpm)	179.9±19.6	172.8±12.1	.03
$HR_{recovery}$ (bpm)	145±16.2	134.3±15.4	<.001
VO_2 max	2173±456.8	2132±498.8	ns
Ve_{max} (L/min)	90.1±21.8	82.4±39	.01
SBP_{rest} (mmHg)	106.7±12.7	102.9±9.2	ns
DBP_{rest} (mmHg)	61.7±10.5	56.7±7.1	.02
SBP_{max} (mmHg)	154.8±26.7	137±5.8	<.001
DBP_{max} (mmHg)	73.7±9.2	69.2±7.3	<.001

SD: standard deviation; DLCO: diffusion lung capacity for carbon monoxide; HR_{max}: heart rate at maximum exercise; $HR_{recovery}$: heart rate in the first 2 min of the recovery phase; HE_{rest}: heart rate at rest; $FEF_{25\%–75\%}$: forced expiratory flow between 25% and 75% vital capacity; FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity; ns: not significant; DBP_{max}: diastolic blood pressure during maximum exercise; DBP_{rest}: diastolic blood pressure at rest; PAS_{max}: systolic blood pressure during maximum exercise; PAS_{rest}: systolic blood pressure at rest; Ve_{max}: minute ventilation at maximum exercise; VO_2 max: the maximum oxygen consumption; % pred: percentage of the predicted value.

in DLCO may be due to changes in pulmonary vascularization innervation. Several mechanisms may explain these changes in pulmonary diffusion. One hypothesis suggests that an alteration in blood flow (less pulmonary vasoconstriction) and, consequently, in pulmonary capillary permeability, would induce changes in the alveolar-capillary membrane. Another possible explanation for this phenomenon involves changes in pulmonary arterial pressure after partial pulmonary sympathetic denervation. These would cause perfusion changes which, together with an increase in bronchomotor tone, would lead to a decrease in CO diffusion.

It is well known that the effect of sympathetic innervation on the heart and systemic vessels is much greater than on the lungs. According to our study, clipping the sympathetic chain mainly affects the cardiovascular system, producing changes in heart rate and blood pressure. These effects in combination are smaller in magnitude and free of clinical repercussions, and are observed both at rest and during exercise. Six months after clipping, we found a reduction in heart rate at peak effort and during recovery, but not at rest. Other authors[8–10] have described that resting heart rate also decreased. This discrepancy could be explained by the use of different surgical techniques or by the size of the sample. The effects of sympathectomy on blood pressure have been well documented. Papa et al.[9] showed a moderate blood pressure response during exercise in subjects who underwent surgery, and Wehrwein et al.[11] found a decrease in systolic pressure, but not in diastolic pressure, 6 months after the procedure. Our results using a different technique are in line with those of the authors mentioned, although we noted some broader effects with regard to the decrease in systolic and diastolic pressures at peak exercise and in systolic pressure at rest. The reduction in heart rate and blood pressure confirms that sympathectomy causes an effect similar to beta-blockers. This decrease in heart rate does not influence VO_2 max, possibly due to a compensatory increase in systolic volume. The reduction in maximum ventilation at peak exercise 6 months after clipping can be explained by an increase in bronchomotor tone, confirmed by the decrease in $FEF_{25\%–75\%}$. We found no changes in VO_2 max, demonstrating that the clipping technique causes minor changes in hemodynamics and lung function, without altering exercise efficiency. We conclude that clipping causes subclinical effects on respiratory function and cardiovascular disease.

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