Impact of phrenic nerve injury on diaphragmatic function after cardiac surgery: a prospective observational study

Dott. MAURIZIO BOTTIROLI (1), Dott. ANDREA DANIELI (2), Dott. RICCARDO PINCIROLI (1)(2), Dott. DANTE FACCHETTI (1), Dott. SIMONE CHECCHI (2), Dott.ssa SARA MASTINU (2), Dott. ANGELO CALINI (1), Dott.ssa MARTA LAZZERI (1), Dott. DANIELE CERIANI (1), Dott. CLAUDIO FRANCESCO RUSSO (1), Prof. GIACOMO BELLANI (2), Dott.ssa MARIA PIA GAGLIARDONE (1), Prof. ROBERTO FUMAGALLI (1)(2)

(1) ASST Grande Ospedale Metropolitano Niguarda, Italia.

(2) Università degli studi di Milano-Bicocca, Italia.

Argomento: Anestesia cardiotoracica

Introduction:

Phrenic Nerve Injury (PNI) is a known complication of cardiac surgery. Aim of this study was to determine its impact on postoperative diaphragmatic function.

Methods:

We designed a prospective-observational study on adult patients undergoing elective open-heart surgery. Electromyography (EMG) was used to study phrenic nerve conduction. Diaphragmatic Compound Muscle Action Potential (CMAP) and latency were measured with chest surface electrodes by cervical electric stimulation preoperatively (T0) and at the first spontaneous breathing trial (T1). Concomitantly, right and left hemidiaphragm excursion was assessed with ultrasonography (US) during Quiet Breathing (QB) and Deep Breathing (DB). An additional US was repeated 7-days after the operation (T2). We defined postoperative PNI as either the absence of a CMAP (paralysis), or a latency higher than 9.75 msec, provided an increase >15% compared to baseline (paresis).

Results:

We enrolled 24 adults (Tab.1). Postoperative PNI could be diagnosed in 13 patients (n=3 paralysis, n=10 paresis). All of the 3 patients with paralysis showed an unilateral injury (2 vs. 1, left vs. right hemidiaphragm, respectively). Among the 10 patients with paresis, 3 had a bilateral injury, while 7 had a one-sided lesion (4 vs. 3, right vs. left). Patients with paralysis showed ipsilateral diaphragmatic immobility at US. Pooling data from all hemidiaphragms (n=48), we observed an overall postoperative reduction in diaphragmatic excursion (Tab.2). Hemidiaphragms affected by ipsilateral PNI showed a higher degree of impairment at US, during both QB and DB, compared to those without EMG alterations (Fig. 1 and 2).

Conclusions:

A high prevalence of PNI after cardiac surgery could be detected in our study, likely of multifactorial origin. A varying degree of severity could be identified, resulting in an abnormal motion of the diaphragm at US. An altered diaphragmatic function due to PNI might lead to postoperative complications, potentially impacting patients' outcome.

Age, years	71 [64 - 76]	
Sex, n (%)	M = 11 (46), F = 13 (54)	
Ejection Fraction, %	60 [51 - 60]	
NYHA class, n	2 [2 - 3]	
EuroSCORE II	2.7 [1,5 - 7,85]	
REDO, n (%)	5 (21)	
CABG, n (%)	3 (13)	
Valve Replacement, (%)	8 (33)	
Combined surgery, n (%)	13 (54)	

Table 1. Baseline characteristic of population (n = 24)

Table 2. Perioperative variations (T0-T1) of diaphragmatic excursion and phrenic nerve function (n=48)

	ТО	T1	p-value
Excursion QB, cm	1,3 [1,2 - 1,8]	1,2 [0,98 - 1,5]	< 0,001
Excursion DB, cm	6,1 [5 - 7,1]	3,4 [2,4 - 4,2]	< 0,001
Latency, msec	7,5 [6,8 - 8,5]	8,7 [7,3 - 10,4]	0,002
CMAP, mV	0,27 [0,18 - 0,42]	0,19 [0,1 - 0,36]	0,008

Figure 1. Postoperative changes in diaphragmatic motion in QB. PNI vs NOPNI

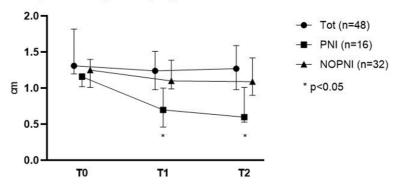


Figure 2. Postoperative changes in diaphragmatic motion in DB. PNI vs NOPNI

