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The Recumbent Position Affects Nasal Resistance: A Systematic Review and Meta-Analysis

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Objective: Nasal diseases are among the main motives for the early discontinuation of continuous positive airway pressure therapy and for long-term therapeutic compliance with mandibular advancement device. Although our clinical experience leads us to the belief that recumbency impacts nasal airflow in some patient populations, there is no consensus regarding the magnitude of this effect and the specific group of patients who are the most affected by this condition. In this study, we conducted a meta-analysis to assess the effect of the recumbent position on nasal resistance and nasal airflow.

Review Methods: PubMed (Medline), Cochrane Library, EMBASE, Scopus, and SciELO databases were checked for relevant studies by two members of the YO-IFOS study group. The two authors extracted the data. The main outcome was expressed as the difference between nasal resistance and nasal airflow before and after recumbency.

Results: Nine studies with a total population of 291 individuals were included in the meta-analysis for nasal resistance after recumbency. We found a statistically significant difference in nasal airway resistance of −0.18 Pa sec/cm^3 as compared to before and after recumbency through rhinomanometry (RMM) analysis. A subgroup analysis revealed a variation of −0.20 Pa sec/cm^3 for patients with snoring or sleep apnea and − 0.10 Pa sec/cm^3 for healthy individuals. Regarding nasal airflow measured with RMM, three studies (n = 32) in asymptomatic controls revealed a statistically significant difference of 47.33 ml/sec.

Conclusions: Recumbency increases nasal resistance and diminishes nasal airflow. This finding is of utmost importance in snorers and sleep apnea patients.

Key Words: Positional rhinitis, sleep apnea, OSA, SAHS.

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INTRODUCTION

The sensation of nasal obstruction when lying down is a frequent complaint among patients in daily otolaryngology practice. This condition was first described and named as "positive posture reaction" by Rundcrantz.[1] Nocturnal nasal congestion, besides being an uncomfortable condition, is a clinically relevant problem due to its association with several disorders, including sleep apnea and asthma.[2]

Although the role of nasal obstruction as an independent cause of sleep apnea is still under discussion,[3] there is no doubt that nasal diseases are among the main motives for the early discontinuation of continuous positive airway pressure (CPAP) therapy.[4] Therefore, nasal diseases should be adequately treated prior to the initiation of CPAP therapy to ensure a long-term adherence.[4] In the same line, there is an association between increased nasal resistance and long-term therapeutic compliance with mandibular advancement device (MAD).[5] Oral breathing was also found to be related with an increased risk of nocturnal asthma attacks.[6] Consequently, as changes in body position can provoke nasal obstruction, they can also trigger asthma attacks.[7] Moreover, the obstruction of the nasal pathway leads to alveolar hyperventilation through the stimulation of the nasopulmonary reflex.[8]

Changes in nasal airway resistance related to recumbency can be measured by several methods. Rhinomanometry (RMM) is currently considered one of the most accepted techniques for the evaluation of nasal ventilation.[9] However, it is primarily used for research purposes as its clinical use is limited by the lack of correlation between the nasal resistance with the clinical symptoms of nasal obstruction. RMM measures the resistance of the transnasal airway; in other words, it estimates how difficult it is to breathe through the nose.

From the Rhinology Study Group of the Young-Otolaryngologists of the International Federations of Otorhinolaryngological Societies (YO-IFOS) (C.C.-HÍQUEZ., C.C.-E., J.R.L., G.C., M.M.-YÁÑEZ., G.M.-C.), Paris, France; Service of Otolaryngology (C.C.-HÍQUEZ., G.M.-C., C.M.-M.), Hospital Complex of Santiago de Compostela, Santiago, Spain; Service of Otolaryngology (C.C.-E.), Donostia University Hospital, San Sebastian, Spain; Foch Hospital (J.R.L.), University of Paris-Saclay, Paris, France; Service of Otolaryngology (M.C.-L.), Hospital Universitario Dr. Peset, Valencia, Spain; Department of Head-Neck Surgery (G.C.), Morgagni-Pierantoni Hospital, Forlì, Italy; Service of Otolaryngology (M.M.-YÁÑEZ.), Hospital Complex of La Coruña, La Coruña, Spain; and the Service of Pneumology (R.A.-P., F.J.G.-B.), Hospital Complex of Santiago de Compostela, Santiago, Spain.

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The nasal resistance is calculated through consecutive measurements of nasal airflow and transnasal pressure. De Vito et al. recommended performing RMM in seated and recumbent body positions and named this assessment as positional rhinomanometry.[10]

Although our clinical experience leads us to the belief that recumbency impacts nasal airflow in some patient populations, there is no consensus regarding the magnitude of this effect and the specific group of patients who are the most affected by this condition. To answer these questions, we carried out this systematic review and meta-analysis to explore the association between supine decubitus position and nasal airflow resistance, without restricting the type of included participants.

METHODS

This review was performed according to preferred reporting items for systematic reviews and meta-analyses (PRISMA) guidelines, and a formal PROSPERO protocol was published according to the National Health Service International Prospective Register of Systematic Review (N° CRD42020212576) prior to the initiation of the study. We also followed the recommendations of the AMSTAR-2 guidelines.[11]

Literature Search: Inclusion and Exclusion Criteria

The criteria for considering studies for this systematic review were based on the population, intervention, comparison, and outcome (PICOS) framework.[12]

- *Participants*: humans.
- *Intervention*: supine recumbency.
- *Comparison*: before and after recumbency, data from uncontrolled studies (quasiexperimental studies), or intervention and no-intervention cohorts from controlled studies (cohorts and clinical trials).
- *Outcomes*: bilateral inspiration nasal airflow and resistance.
- *Types of studies*: clinical trials, case series, and prospective and retrospective cohort studies published in peer-reviewed journals. We did not include case reports, theses, or meetings' communications. There were no restrictions by date or publication type. The search was last updated in December 2019. We included studies published in English, Spanish, German, French, Italian, and Portuguese.
- *Exclusion criteria*: 1) studies carried out on syndromic patients; 2) duplicated publications; 3) unilateral data; and 4) use of nasal decongestants, as they were shown to annul the postural effect.[13]

Search Strategy

We followed the recommendations of the PRISMA statement to perform a systematic review and searched the following databases: PubMed (Medline), Cochrane Library, EMBASE, Scopus, ScienceDirect, and SciELO. We used a predefined search strategy that is described in Supporting Data 1. The abstracts of the retrieved papers were thoroughly reviewed by two authors (C.C.H. and C.C.E.), and the publications that could potentially fulfill the inclusion criteria were selected for a full-text review. In case of discrepancies between reviewers regarding the selection of the abstracts, the corresponding papers were included in the full-text review stage for a final assessment. We also manually reviewed the references of all selected articles to identify any potentially missing publication.

Data Extraction and Analysis

Two authors (C.C.H. and C.C.E.) independently analyzed the articles that met the inclusion criteria and extracted the relevant data. Extracted variables encompassed: sample size; age; initial diagnosis; confounding factors including body mass index, prevalence of allergy, use of nasal steroids, and smoking habits; methods used to measure the nasal resistance of airflow; and the main outcome. The main outcome was expressed as percentage change in nasal airflow and in nasal resistance before and after recumbency along with its 95% confidence interval. Flow and resistance were expressed in cm^3/sec and Pa sec/cm^3, respectively. All the data are recorded in Table I.

Quality Assessment

We assessed the selected articles for both: the level of evidence and the quality. The level of evidence was classified according to the Oxford Centre for Evidence-Based Medicine Levels (2011).

The risk of bias was assessed according to the quality assessment of quantitative studies checklist from the National Institute for Health and Clinical Excellence.[14] The assessed items are described in Table II. The items 2.1, 2.3, 2.6, 2.10, 3.5, 3.6, and 4.2 of the checklist were not assessed as they were not applicable to the selected studies.

Statistical Analysis

Data were analyzed with STATA for Macintosh v. 15.1 (StataCorp®). P values < .05 were considered statistically significant. Data were presented as 95% confidence interval.

We used Cochrane Collaboration's Review Manager Software (REVMAN), version 5.4 (Nordic Cochrane Centre, Cochrane Collaboration, 2020, Copenhagen, Denmark), to conduct the meta-analysis. The heterogeneity was checked using the Q-test and I^2 test. A fixed effects model was used for $I^2 < 50\%$, and a random effects model was adopted when I^2 was $\geq 50\%$. Finally, the publication bias was assessed using the funnel plot and Egger regression.

RESULTS

Search Results

A flow chart of the search process is shown in Figure 1. The initial search retrieved 298 publications. After checking all titles and abstracts, 33 studies were selected for full-text review. A total of 15 studies with a total population of 535 patients met the inclusion criteria.

The 18 publications were excluded after full-text review because of the following reasons: 11 studies encompassed RMM tests but the results were not reported; four studies did not assess bilateral RMM; two studies applied nasal decongestant; and one study did not evaluate the change in nasal resistance between seating and recumbency positions.

Results of the Included Studies

A summary of the selected studies is represented in Table I.

General results. The age ranged from 25.8 to 55 years. The lowest mean age corresponds to the study published by Hiyama et al. (25.8 years), and the highest

TABLE I.
Description of the Included Studies.

Author (yr)	Design/Level of Evidence	Method	Sample Size and Sex	Age (yrs) Mean ± SD (range)	Initial Diagnosis	Technique	Main Outcome (Difference ± SD)		% of Difference (CI 95%)	Confounding Factors. Mean ± SD (Range)
Karlsson et al. 2020	Open nonrandomized clinical trial Level 2	Adaptation: NR Order: sitting–recumbent Time to change: 10 min Degree of decubitus: 0°	41 (16 M, 25 F)	33.0 ± 10.0 (NR)	Chronic nasal obstruction	Anterior active RMM. Nasal resistance ($Pa\ sec/cm^3$)	0.38 ± 0.47	0.33 ± 0.10	−13.16 (−51.87 ± 22.55)	BMI: 25.4 ± 3.7 Allergy: 16 (39%) Smokers: 7 (17.1%) Nasal steroids: 22 (53.7%)
Huang et al. 2020	Open nonrandomized clinical trial Level 2	Adaptation: NR Order: sitting–recumbent Time to change: 5 min Degree of decubitus: 0°	30 (22 M, 8 F)	46.7 ± 13.0 (NR)	Mild OSA (AHI 5–14)	Anterior active RMM. Nasal resistance ($Pa\ sec/cm^3$)	0.19 ± 0.67	0.26 ± 0.21	33.85 (−7.35 ± 75.06)	BMI: 24.8 ± 3.0 Allergy: 0 Smokers: NR Nasal steroids: NR
						Posterior active RMM. Nasal resistance. ($Pa\ sec/cm^3$)	0.24 ± 0.19	0.29 ± 0.15	17.62 (−18.34 ± 53.59)	
			32 (27 M, 5 F)	47.9 ± 8.3 (NR)	Moderate–severe OSA (AHI ≥15)	Anterior active RMM. Nasal resistance ($Pa\ sec/cm^3$)	0.20 ± 0.15	0.30 ± 0.30	47.26 (−10.86 ± 105.30)	BMI: 28.2 ± 4.0 Allergy: 0 Smokers: NR Nasal steroids: NR
						Posterior active RMM. Nasal resistance ($Pa\ sec/cm^3$)	0.32 ± 0.26	0.43 ± 0.34	32.81 (−13.72 ± 79.35)	
Zeng et al. 2008	Open nonrandomized clinical trial Level 2	Adaptation: NR Order: sitting–recumbent Time to change: NR Degree of decubitus: NR	26 (21 M, 5 F)	50.9 ± 2.2 (NR)	MAD responders	Posterior active RMM. Nasal resistance at airflow 500 ml/sec ($Pa\ sec/cm^3$)	0.64 ± 0.05	1.05 ± 0.12	64.62 (56.93–72.30)	BMI: 28.7 ± 0.8 Allergy: 0 Smokers: NR Nasal steroids: NR
			12 (8 M, 4 F)	55.0 ± 2.1 (NR)	MAD non-responders		0.92 ± 0.10	1.16 ± 0.15	25.53 (14.68–36.38)	BMI: 34.3 ± 1.1 Allergy: 0 Smokers: NR Nasal steroids: NR
Duggan et al. 2004	Open nonrandomized clinical trial Level 2	Adaptation: NR Order: sitting–recumbent Time to change: NR Degree of decubitus: 0°	10 (5 M, 5 F)	33.5 ± NR (23–58)	Asymptomatic controls	Forced oscillation technique. Nasal resistance ($Pa\ sec/cm^3$)	0.21 ± 0.08	0.26 ± 0.11	28.57 (−11.57–68.72)	BMI: NR Allergy: 0 Smokers: 0 Nasal steroids: NR
			17 (10 M, 7 F)	53.54 ± NR (23–80)	Asthmatic patients		0.34 ± 0.32	0.48 ± 0.36	42.53 (–25.53; 110.50)	BMI: NR Allergy: 10 Smokers: 0 Nasal steroids: NR
Virkkula et al. 2003	Open nonrandomized clinical trial. Level 2	Adaptation: NR Order: sitting–recumbent Time to change: 5 min Degree of decubitus: NR	41 (41 M, 0 F)	41 ± 8.9 (26–62)	Suspected OSA	Anterior RMM at radius 200. Nasal resistance ($Pa\ sec/cm^3$)	0.28 ± 0.20	0.37 ± 0.36	31.56 (−13.07–76.19)	BMI:28.1 ± 3.5 (NR) Allergy: 6 (15%) Smokers: 20 (49%) Nasal steroids: 3
			19 (14 M, 5 F)	42 ± NR (31–70)	Asymptomatic controls		0.16 ± 0.07	0.23 ± 0.14	43.75 (0.51–86.99)	BMI:28.1 ± NR (22-37) Allergy: 8 (42%) Smokers: 1 (49%) Nasal steroids: 1

Hiyama et al. 2002	Quasiexperimental study Level 4	Adaptation: NR Order: sitting–recumbent Time to change: 10 min Degree of decubitus: 0°	15 (7 M, 8 F)	25.8 ± 1.2 (NR)	Asymptomatic controls	Anterior active RMM. Nasal patency ratio. Nasal resistance (units NR)	65.9 ± 21.1	48.0 ± 17.8	−27.16 (−48.36 to −5.96)	BMI: 21 ± 2.9 (NR) Allergy: 0 Smokers: NR Nasal steroids: NR
De Vito et al. 2000	Quasiexperimental study Level 4	Adaptation: NR Order: sitting–recumbent Time to change: NR Degree of decubitus: NR	36 (31 M, 5 F)	52 ± 9.72 (34–79)	OSA, retropalatal collapse	Anterior active RMM. Nasal resistance (Pa sec/cm^3)	0.34 ± 0.13	0.54 ± 0.34	58.11 (23.85–93.80)	BMI: 33.1 ± 7.62 (20.4–46.4) Allergy: NR Smokers: NR Nasal steroids: NR
Stroud et al. 1999	Open nonrandomized clinical trial Level 2	Adaptation: NR Order: sitting–recumbent. Time to change: 15 min Degree of decubitus: 0°	27 (NR) 20 (NR)	NR ± NR (NR)	Patients complaining of nocturnal nasal congestion Asymptomatic controls	Anterior active RMM at 150 Pa. Nasal resistance (Pa sec/cm^3)	0.16 ± NR 0.17 ± NR	0.25 ± NR 0.17 ± NR	61.15 (NR) 2.99 (NR)	BMI: NR Allergy: 29 Smokers: 15 Nasal steroids: 6
Desfonds et al. 1998	Open nonrandomized clinical trial. Level 2	Adaptation: NR Order: sitting–recumbent Time to change: 10 min Degree of decubitus: 0°	70 (63 M, 7 F) 11 (6 M, 5 F)	52 ± 1.4 (NR) 30 ± 5 (NR)	Snorers Asymptomatic controls	Posterior active RMM. Nasal resistance at 500 ml/sec	No individual data reported. SSD No individual data reported. SSD		182 (160.94–203.36) 128 (114.87–141.13)	BMI: 29 ± 0.8 (NR) Allergy: NR Smokers: NR Nasal steroids: NR BMI: NR Allergy: 0 Smokers: NR Nasal steroids: 0
Tvinnereim et al. 1996	Quasiexperimental study Level 4	Adaptation: NR Order: sitting–recumbent Time to change: NR Degree of decubitus: NR	12 (12 M, 0 F)	NR ± NR (33–75)	Asymptomatic controls	Posterior active RMM with body plethysmograph. Nasal resistance (Pa sec/cm^3) Posterior active RMM with body plethysmograph. Nasal airflow (ml/sec)	0.15 ± 0.07 249 ± 89	0.29 ± 0.18 208 ± 53	93.33 (20.48–166.18) −16.47 (−40.00 to 7.07)	BMI: NR Allergy: 0 Smokers: NR Nasal steroids: 0
Altissimi et al. 1996	Open nonrandomized clinical trial. Level 2	Adaptation: 30 Order: sitting–recumbent Time to change: 3 min Degree of decubitus: 20°	40 (NR) 30 (NR) 25 (NR)	NR ± NR (NR)	Asymptomatic controls Allergic rhinitis Vasomotor rhinitis	Anterior active RMM. Nasal resistance at 150 Pa.	No individual data reported. No individual data reported. No individual data reported.		29 (−47.44 to 105.44) 16 (−180 to 212) 65 (−203.52 to 333.52)	BMI: NR Allergy: NA Smokers: NR Nasal steroids: 0
Miljeteig et al. 1995	Quasiexperimental study Level 4	Adaptation: 30 min Order: sitting–recumbent Time to change: 5 min Degree of decubitus: 0°	21 (21 M, 0 F)	40 ± 13 (22–76)	Asymptomatic controls	Anterior active RMM. Nasal resistance (Pa sec/cm^3)	0.14 ± 0.07	0.35 ± 0.32	150 (49.93 to 250.07)	BMI: NR Allergy: 0 Smokers: NR Nasal steroids: NR

(Continues)

TABLE I.
Continued

Author (yr)	Design/Level of Evidence	Method	Sample Size and Sex	Age (yrs) Mean ± SD (range)	Initial Diagnosis	Technique	Main Outcome (Difference ± SD)		% of Difference (CI 95%)	Confounding Factors. Mean ± SD (Range)
Riechelman et al. 1994	Quasiexperimental study Level 4	Adaptation: NR Order: sitting–recumbent Time to change: NR Degree of decubitus: 0°	15 (NR)	NR ± NR (NR)	Asymptomatic controls	Anterior active RMM. Airflow at 150 Pa (ml/sec)	362 ± 166	350 ± 167	–3.31 (–36.23 to 29.60)	BMI: NR Allergy: 0 Smokers: NR Nasal steroids: NR
Rao et al. 1970	Quasiexperimental study Level 4	Adaptation: 10–15 min Order: recumbent–sitting Time to change: 10 min Degree of decubitus: 0°	5 (5 M, 0 F)	NR ± NR (19–49)	Asymptomatic controls	Nasal spirometry. Nasal airflow (ml/sec).	223.33 ± 48.33	163.33 ± 38.33	–26.87 (–51.08 to –2.66)	BMI: NR Allergy: 0 Smokers: NR Nasal steroids: NR
Rundcrantz 1969	Open nonrandomized clinical trial. Level 2	Adaptation: NR Order: sitting–recumbent Time to change: 2 min Degree of decubitus: 0°	17 (NR) 18 (NR)	NR ± NR (14–41)	Asymptomatic controls Rhinitis (allergic and infectious rhinitis)	Posterior active RMM. Nasal resistance at 500 ml/sec. (Pa sec/cm^3)	0.14 ± 0.04 0.29 ± 0.14	0.24 ± 0.08 0.88 ± 0.44	76.47 (44.88–108.06) 178.92 (103.84; 254.00)	BMI: NR Allergy: NA Smokers: NR Nasal steroids: 0

AHI = apnea-hypopnea index; BMI = body mass index; F = female; M = male; MAD = mandibular advancement device; NA = not applicable; NR = not reported; OSA = obstructive sleep apnea; RMM = rhinomanometry; SD = standard deviation.

TABLE II.
Assessment of the Risk of Bias (NICE guidelines, Appendix F).

	Karlsson	Huang	Zeng	Duggan	Virkkula	Hiyama	De Vito	Stroud	Desfonds	Tvinnereim	Altissimi	Miljeteig	Riechelman	Rao	Rundcrantz
1.1	+	+	+	+	+	+	+	+	+	+	–	+	+	+	+
1.2	++	++	++	+	++	++	++	++	++	++	NR	NR	NR	+	NR
1.3	+	+	+	+	+	–	+	+	+	–	NR	NR	NR	–	NR
2.2	++	++	+	++	++	++	+	++	++	+	++	++	++	++	++
2.4	–	NA	–	–	–	NA	NA	–	–	NA	–	NA	NA	NA	–
2.5	++	++	+	+	+	++	+	++	++	+	+	+	+	++	+
2.7	++	NA	++	++	++	NA	NA	++	++	NA	++	NA	NA	NA	++
2.8	–	++	++	++	++	++	++	++	–	++	++	++	++	++	++
2.9	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
3.1	+	++	++	+	++	++	++	++	++	+	++	++	++	+	+
3.2	+	+	++	++	++	++	++	++	++	++	++	++	++	++	++
3.3	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
3.4	+	++	++	+	++	+	++	++	++	+	+	++	+	++	++
4.1	+	++	++	–	+	++	++	+	+	++	+	++	++	++	++
4.3	NR	NR	++	NR	NR	++	++	NR	+	++	NR	++	NR	++	++
4.4	++	++	++	++	++	++	++	–	–	++	–	++	++	++	++
4.5	++	++	++	++	++	++	++	–	–	++	–	++	++	++	++
4.6	+	+	++	+	+	+	+	–	–	+	–	+	+	+	++
5.1	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
5.2	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

1.1 Is the source population or source area well described?; 1.2 Is the eligible population or area representative of the source population or area?; 1.3 Do the selected participants or areas represent the eligible population or area?; 2.2 Were interventions well described and appropriate?; 2.4 Were participants or investigators blind to exposure and comparison?; 2.5 Was the exposure to the intervention and comparison adequate?; 2.7 Were other interventions similar in both groups?; 2.8 Were all participants accounted for at study conclusion?; 2.9 Did the setting reflect usual practice?; 3.1 Were outcome measures reliable?; 3.2 Were all outcome measurements complete?; 3.3 Were all important outcomes assessed?; 3.4 Were outcomes relevant?; 4.1 Were exposure and comparison groups similar at baseline? If not, were these adjusted?; 4.3 Was the study sufficiently powered to detect an intervention effect (if one exists)?; 4.4 Were the estimates of effect size given or calculable?; 4.5 Were the analytical methods appropriate?; 4.6 Was the precision of intervention effects given or calculable? Were they meaningful?; 5.1 Are the study results internally valid (i.e., unbiased)?; 5.2 Are the findings generalizable to the source population (i.e., externally valid)?

NA = not applicable; NR = not reported.

mean age was reported by Zeng et al. in a cohort of nonresponders to MAD therapy (55 years).

The mean sample size was 28.5. The largest study was performed by Desfonds and colleagues in a cohort of snorers (70 patients),[13] and the smallest study was that of Rao et al. (five patients).[15]

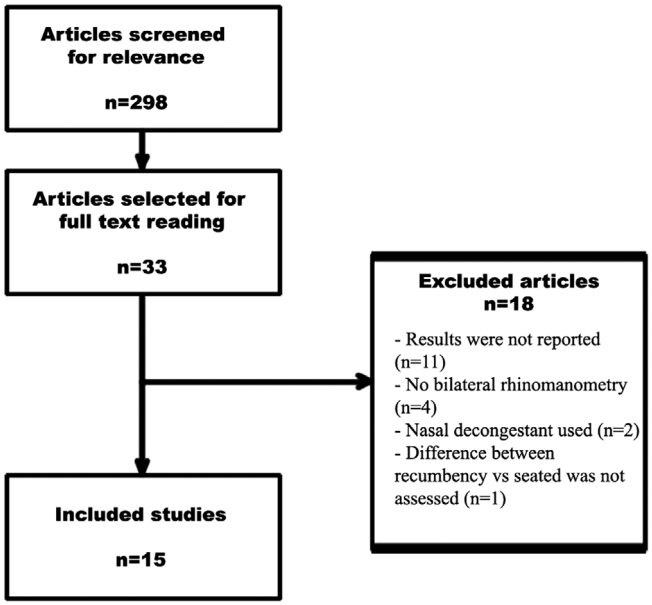

Fig. 1. Flow chart of the study selection process following the PRISMA guidelines.

Nasal airway resistance. Thirteen of the 15 localized studies had explored nasal resistance after recumbency.[1,2,5,7,10,13,16–22] All of the 13 studies, with the exception of Karlsson et al., found that nasal resistance increases with decubitus.

Nine studies with a total sample size of 332 individuals were included in the meta-analysis (Fig. 2). The pooled effect of the random model showed a statistically significant difference in nasal airway resistance of −0.17 Pa sec/cm^3 between the readings before and after recumbency. To get a perspective of this results, note that a measurement of 0.30 Pa sec/cm^3 is usually considered the upper normal limit of the nasal resistance.[23]

Four studies (n = 177) encompassed patients with snoring or sleep apnea. Under a random effect model, the pooled effect yielded a statistically significant difference in nasal airway resistance of −0.20 Pa s/cm^3.

Finally, five studies were carried out in healthy individuals, exclusively. Restricting the analysis to these studies showed a significant difference in nasal airway resistance of −0.10 Pa sec/cm^3.

Two studies investigated the change in nasal resistance in patients with rhinitis.[1,18] Rundcrantz found a statistically significant difference in nasal airway resistance between the readings before and after recumbency,[1] whereas Altissimi et al. did not find this change.[18] It is worth noting that the study by Karlsson et al. is not included as they performed

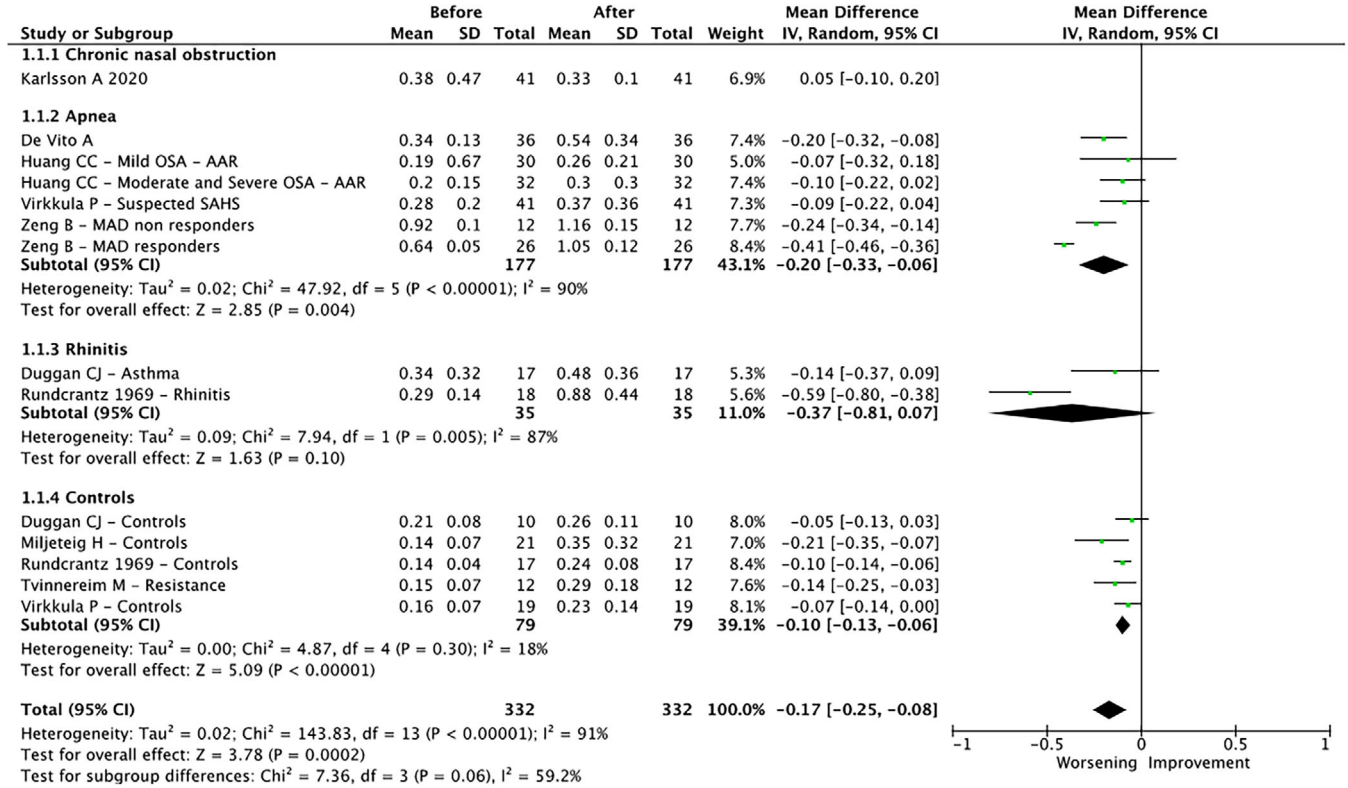

Fig. 2. Forest plot. Difference in nasal resistance after recumbency (in Pa sec/cm^3) in the selected studies including the analyzed subgroups (apnea patients, rhinitis, and healthy controls). [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

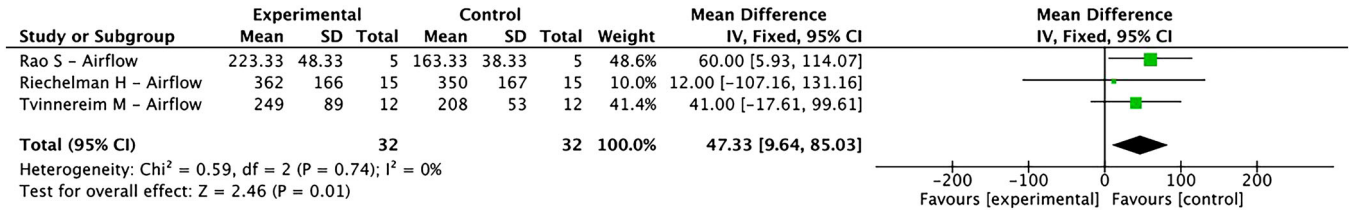

Fig. 3. Forest plot. Difference in nasal airflow after recumbency (in cm^3/sec) for all included studies. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

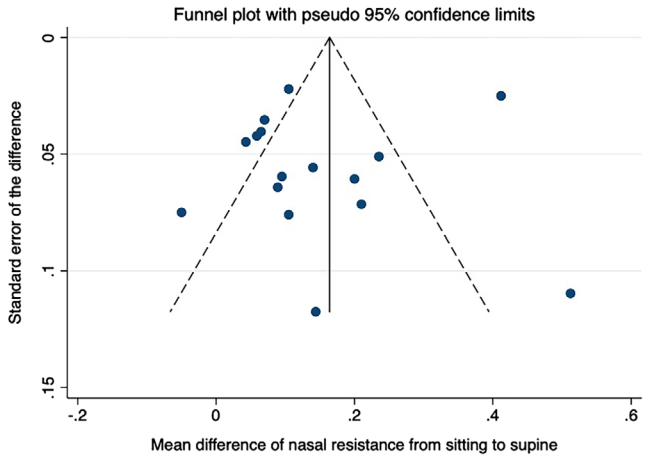

Fig. 4. Funnel plot assessing the risk of publication bias for the studies included in the meta-analysis of the nasal resistance. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

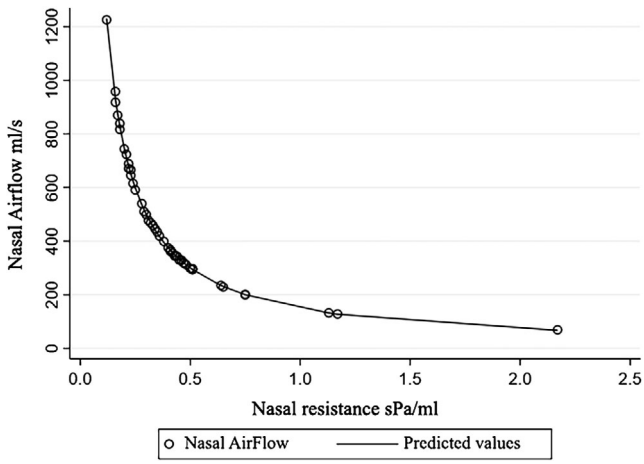

Fig. 5. Exponential relationship between nasal airflow (y axis) and nasal resistance (x axis).

their study in patients complaining of chronic nasal obstruction, but without a specific diagnosis.[21]

Only one study explored the difference in nasal resistance in asthmatic patients and reported a statistically significant difference between before and after recumbency measurements as compared with controls.[7]

Nasal airflow. A total of three studies (n = 32) in asymptomatic controls assessed the change in nasal airflow after recumbency.[15,16,24] The findings of these studies were combined in a meta-analysis (Fig. 3), and the pooled effect revealed a statistically significant difference of 47.33 ml/sec under a fixed effects model. Data for nasal airflow are less standardized; however, 800 ml/sec is usually considered the lower normal limit.[25]

Controlled studies. There were six controlled studies[1,2,7,13,18,22]; however, as they include non-comparable cohorts, the results of these studies could not be meta-analyzed. The controls were compared with apnea patients in the study of Virkulla et al.,[22] with patients complaining from nocturnal nasal congestion in the study of Stroud et al.,[2] with patients suffering from rhinitis in the studies of Rundcrantz[1] and Altissimi et al.,[18] with snorers in the study of Desfonds et al.,[13] and finally with asthmatic patients in the study of Duggan et al.[7]

Publication Bias and Small Study Bias

The funnel plot (Fig. 4) and Egger regression test indicate the absence of publication bias ($t = -0.67$; $P = .51$).

DISCUSSION

This is the first systematic review and meta-analysis that assesses the effect of recumbency on nasal airflow and nasal resistance.

We revealed two main findings. First, recumbency increases nasal resistance and diminishes nasal airflow in the whole studied population. Second, the effect on nasal resistance varies between individuals, as it is stronger in apnea patients (0.20 Pa sec/cm^3) than in controls (0.10 Pa sec/cm^3). This effect also seems to be more evident in rhinitis and asthmatic patients than in other individuals, but the findings of the corresponding studies could not be pooled in a meta-analysis.

This finding is clinically relevant as, despite some existing debates, the upper normal limit for the nasal resistance is 0.30 Pa sec/cm^3[26]; therefore, a change of 0.20 Pa sec/cm^3 represents a remarkable indicator in the worsening of the nasal resistance. The relationship between nasal resistance and nasal airflow follows an exponential curve because nasal resistance is mathematically calculated from an exponential value of the nasal volume and from the differential nasal pressure (Fig. 5).[27] It means that, in patients suffering from severe nasal

congestion, small variations in nasal resistance do not lead to significant changes in nasal airflow. However, in patients with nasal resistance values that range between 0.2 Pa sec/cm^3 and 0.5 Pa sec/cm^3 (upper normal limits),[26] even a small variation in nasal resistance can induce huge changes in nasal airflow. Therefore, in this group of borderline patients, small variations in nasal resistance like those found in this meta-analysis may cause nocturnal nasal congestion in asymptomatic patients. In fact, Stroud et al. found that patients with nocturnal nasal congestion had normal basal nasal resistance (0.16 Pa sec/cm^3) that subsequently increased to the limit of normality (0.3 Pa s/cm^3) after recumbency, providing therefore a sufficient evidence to consider this change as potentially clinically relevant.[2]

There would be a great interest in knowing if those differences in airflow and nasal resistance are clinically relevant. This means that if those differences are detected by the subjects under study or if they could change the mode of breathing. However, little information has been published in this regard. The number of patients who changed from normal to pathologic nasal resistance was only determined by De Vito et al., who studied patients suffering from sleep apnea. De Vito et al. found that 31% of patients with normal basal nasal resistance developed pathologic nasal resistance upon recumbency.[10] This study applied the limit 0.50 Pa sec/cm^3, revealing therefore that the percentage of patients who developed pathologic nasal resistance could have been even higher if the standard limit of 0.30 Pa sec/cm^3 were used.

This finding is of particular importance in patients for whom nocturnal nasal breathing is crucial. This group of patients includes individuals with sleep apnea and asthmatics. Only Solomon reported that several participants begun mouth breathing within seconds after becoming supine.[28] However, none of the other studies investigated this outcome. On the contrary, some studies reported the number of patients who did not have any change in nasal resistance. This frequency was 13.33% and 9.52% as reported by Hasegawa et al.[29] and Miljeteig et al.,[19] respectively. Furthermore, there is a remarkable interindividual variation, which might range from 20% to 120% according to Hiyama et al.[17] It means that nocturnal nasal congestion is not a universal disorder; therefore, there must be other factors contributing to the effect of recumbency on nasal resistance apart from the change in body posture.

The underlying physiology behind nocturnal nasal congestion is not clear. Three disorders were suggested to play a role in the occurrence of nocturnal nasal congestion, and the event can be an outcome of the combined effect of the three disorders.

The first hypothesis is related to venous stasis syndrome. This hypothesis is supported by the fact that in the erect position, the pressure within the jugular vein is almost zero, while it increases from 4.5 mmHg to 11 mmHg with the 0° decubitus.[30] In line of this, nasal resistance also increases after compression of the jugular vein.[1]

The second hypothesis deals with baroreceptor-mediated reflex. Previous studies demonstrated that the application of unilateral pressure to the axillary region,[31] or in the lateral side of the body,[15] provokes an ipsilateral nasal congestion and contralateral decongestion, suggesting therefore that a neural reflex is mediated by pressure receptors.

The third hypothesis suggests that preponderance of the parasympathetic autonomous system increases with decubitus.[29] This hypothesis is based on findings of animal studies in which electrical stimulation of the cat's cervical sympathetic trunk yielded a greater reduction in blood flow.[32] Electrical stimulation of the vagus nerve in pigs also caused bilateral vasodilatation of the nasal mucosa.[33] The effect of body position on nasal permeability is provoked by a reflex arc that is controlled by efferent nerve fibers extending from the cervical sympathetic plexus and by afferent fibers projecting from pressure receptors in the subcutaneous tissue. In humans, Riechelman et al. studied the effect of body position on nasal resistance, with alfa 1 and alfa 2 adrenergic receptors blockers, and reported an increased positional effect under alfa 1 blocking. Riechelman et al. concluded that alfa 1 adrenergic receptors are the main regulators of the filling pressure of capacitance vessels of the nose.[24]

Despite that data regarding the worsening in nasal resistance with decubitus are unanimous, there is some concerns regarding the role of some unstudied potential confounding variables. The first concern is that nasal resistance is not always constant in healthy individuals. It is influenced by several factors such as positional changes, menstrual cycle, partial pressures of oxygen and carbon dioxide (breath holding or hyperventilation), temperature, humidity, or exercise. Therefore, RMM has been criticized by some authors. However, measuring the difference of nasal resistance according to body position under equal conditions allows to neutralizing the effect of potential confounding factors.

On the other side, several factors such as smoking habit, obesity, rhinitis, or drug intake may influence the magnitude of the effect of body position on nasal resistance.[34]

Smoking damages the nasal mucosa.[35] Stroud et al. found that smokers had a significantly greater posture effect compared with nonsmokers or with patients without rhinitis. In fact, this difference was six times higher for smokers.[2] Unfortunately, none of the studies included in this meta-analysis analyzed the effect of body position in a subgroup of smokers.

Although we could not perform a subgroup analysis limited to patients with rhinitis, the presence of rhinitis seems to be clearly related to an increased positional effect.[1,18,29] Rundcrantz found that the positional effect in patients with rhinitis was three times higher than that in asymptomatic controls.[1] The magnitude of the effect reported by Stroud and colleagues was even higher as they found that the difference in nasal resistance in patients with rhinitis was 23 times higher than that in controls.[2] On the contrary, Virkkula et al. found that nasal resistance did not increase in 15% of the patients with history of allergic rhinitis.[22] Interestingly, Rundcrantz found that eight out of nine patients with positional increased nasal resistance showed normal results in RMM after receiving a 6-month treatment with subcutaneous injections of

allergen.[36] Several studies mentioned the number of patients with allergy; however, only few of these studies performed a subgroup analysis to control for this confounding factor.

Nasal steroids have been shown to normalize the postural effect in rhinitis patients, with an increased normalizing effect after 2 weeks.[29] Nevertheless, most studies did not control for this variable, which could have introduced some bias.

Finally, the degree of decubitus is an important variable reported by several authors.[1,2,17,29] Hasegawa et al. found that the nasal resistance increases in a linear relationship in controls and exponentially in allergic rhinitis patients with an increasing degree of decubitus.[29] Rundcrantz also found a progressive worsening of nasal resistance with increased decubitus using the critical point 20°.[1] Most authors explored participants under 0° of decubitus; however, others did not report this variable, which might influence the final results.

Knowledge Gap

There are some knowledge gaps, which could guide researchers to future studies. None of the studies were carried out in children. This fact is of utmost importance that according to Mew's tropic premise, keeping the mouth closed is fundamental for a proper facial growth.[37]

There is a lack of information regarding the duration of increased nasal resistance caused by decubitus. Clinicians are concerned about nasal resistance during the whole night, not only during the first few minutes. If the positional effect were reversible after time, our concern would change; however, the available related data are limited. Kurita and colleagues performed 15 repeated measurements of change in nasal resistance after 5 minutes of sitting and after 5 minutes in supine posture. They found an almost immediate worsening with supine position, but nasal resistance decreased gradually during the next few minutes.[38] Similar findings were reported by Broms et al. who explored nasal resistance for 90 minutes of sitting and 30 minutes in recumbent position. Broms et al. found a respective progressive improvement and worsening.[39] However, they did not inquire into this finding, which we think that it is of utmost importance.

There is only one study that assessed nasal resistance before and after turbinate surgery, normalizing therefore the positional effect by comparing it to controls.[40] There is a crucial need for further studies that investigate the effect of turbinate surgery, as it could be a treatment for patients who snore or have sleep apnea.

Limitations

As with any systematic review, we may have missed some studies in the literature despite doing our best to provide a comprehensive review. Furthermore, conclusions drawn from systematic reviews and meta-analyses depend on the quality of the included studies. The quality of the studies included in our review was poor regarding the adjustment for confounding factors. The conclusions can only be applied to the studied population (probably males in middle age) and cannot be generalized to older individuals or children due to lack of data as discussed earlier. RMM has shown a poor correlation with clinical symptoms. Therefore, it would be interesting to know the clinical relevance of the observed changes in this technique. However, none of the included studies had explored this variable. Finally, although some studies were carried out in asymptomatic healthy individuals, it is still not clear what is meant by the used term "normal" is and what was the cut-off value after which the worsening in nasal resistance and nasal airflow became evident to the patient. Furthermore, the estimated confidence interval was very wide due to the small sample size.

CONCLUSION

Recumbency increases nasal resistance and diminishes nasal airflow. This finding is of outmost importance in snorers and sleep apnea patients. However, additional research is needed to correlate and understand the clinical implications of the changes seen on the objective measurements. Furthermore, there is a clear need for studies in subgroup populations such as sleep apnea, rhinitis, or asthmatic patients to expand the available sample size.

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