The Journal of Laryngology and Otology
June 1991, Vol. **105**, pp. 436–438

Effect of enlarged adenoids on arterial blood gases in children

MOHAMMED S. KHALIFA, M.D., REDA H. KAMEL, M.D., MONA ABU ZIKRY, M.D.,
TAREK M. KANDIL, M. S. (Cairo, Egypt)

Abstract
The enlarged adenoid is a common disorder in children resulting in nasopharyngeal obstruction. Many authors suggest that increased nasal resistance to respiration may cause disturbances in the pulmonary ventilation and carry the risk of obstructive sleep apnoea and/or cardiopulmonary syndrome.

This study comprised 30 children complaining of long-standing nasal obstruction due to enlarged adenoids. Adenoidectomy was performed and the arterial blood gases were measured before and one month after surgery. Twelve normal children were also included as controls. Statistical evaluation of the results showed that O_2 saturation and arterial O_2 tension (PaO_2) were significantly low before the operation, and increased significantly after surgery. Arterial CO_2 tension ($PaCO_2$) was insignificantly low before operation, but decreased significantly after adenoidectomy. It was concluded that enlarged adenoid may be associated with ventilatory impairment which is reversible after adenoidectomy.

Introduction

The adenoid occupies a key position in the development of the immune process. It is minimal in size at birth, increases in size at one to three years of age when active immunity is being established and may recede at puberty (Belenky, 1983). The most common symptom of adenoid enlargement is nasal obstruction, with resultant mouth breathing and/or snoring. This may result in sleep apnoea and/or cardiopulmonary syndrome (Menashe *et al.*, 1965).

Although many authors have studied the role of adenotonsillar enlargement on pulmonary ventilation (Menashe *et al.*, 1965; Cayler *et al.*, 1969; Macartney *et al.*, 1969; Talaat and Nahhas, 1983; Sofer *et al.*, 1988), very few have considered the role of the enlarged adenoid alone (Luke *et al.*, 1966; Yonkers and Spaur, 1987). It was also noticed that all those researches which studied the adenoid alone comprised few cases. The aim of this study was to include more cases of adenoid enlargement and study its effect on pulmonary ventilation.

Material and methods

This study comprised 30 children complaining of long-standing nasal obstruction due to enlarged adenoids. They were selected from the outpatient clinic of the Abu-El-Reesh Paediatric Hospital, Cairo University, in the period from January, 1988 to May, 1989. Their ages ranged between three and 11 years with a mean of seven years. There were 17 males and 13 females. To exclude the role of the tonsils on the results, four patients had had their tonsils removed at least one year before this study and in the rest of the patients, the tonsils were normal in size and causing no obstruction of the airway and there was no indication for tonsillectomy. Patients who had any other associated cause of nasal obstruction were excluded. Twelve normal children were also included as control. Their ages ranged between 3 and 11 years with a mean of 6.5 years. There were seven males and five females. They were free from adenoid, tonsils or any cause of nasal obstruction.

Arterial blood gases were measured for the patients and control group using the ABL2 Blood Gas Analyzer. It is well known that blood freely flowing from a warmed capillary bed corresponds well to arterial blood. In this study, because it was difficult to get arterial samples from the children, capillary tubes were used to obtain arterialized capillary blood. The samples obtained were from the tips of the index finger after submerging the hand in hot water (40–43°C).

Adenoidectomy was performed under general anaesthesia. The arterial blood gases were remeasured one month after adenoidectomy. Although 50 patients were operated upon, 20 patients were lost to follow-up.

In comparing the results of arterial blood gases in the patients before and after adenoidectomy, the paired t-test was used. In comparing the results of arterial blood gases in the patients before adenoidectomy with their control, the unpaired t-test was used. In comparing the results of arterial blood gases in the patients after surgery with their control, the unpaired t-test was used.

[1]normal value = 95–98 per cent.
[2]normal value = 75– mm Hg.
[3]normal value = 32–42 mm Hg.
Accepted for publication: 23 February 1991.

Results

Statistical evaluation of the results of the arterial blood gases in the patients before and after adenoidectomy showed that: before surgery O_2 saturation ranged between 84–94 per cent[1] with a mean of 89.4, SD 3.069 per cent. One month after surgery, O_2 saturation ranged between 87–97 per cent with a mean of 93.5, SD 2.99 per cent. There was a statistically significant increase in the O_2 saturation after adenoidectomy (O_2 saturation mean before—after = 4.117, SD 3.71, $p<0.0001$). Before surgery PaO_2 ranged between 57–84 mmHg[2] with a mean of 70.933, SD 6.068 mmHg. One month after surgery, PaO_2 ranged between 67–102 mmHg with a mean of 87.833, SD 12.132 mmHg. There was a statistically significant increase in the PaO_2 after adenoidectomy (PaO_2 mean before–after = 16.9, SD 11.48, $p<0.0001$). Before surgery $PaCO_2$ ranged between 32–44 mmHg[3] with a mean of 38.567, SD 3.54 mmHg. After adenoidectomy, $PaCO_2$ ranged between 33–44 mmHg with a mean of 37.6, SD 3.136 mmHg. There was a statistically significant decrease in the $PaCO_2$ after adenoidectomy ($PaCO_2$ mean before—after = 0.97, SD 2.28, $p<0.0324$).

The comparison between the patients before surgery with the control group as regards the arterial blood gases, showed that the mean O_2 saturation was 89.4, SD 3.069 per cent. The mean O_2 saturation for the control group was 95.917, SD 1.443 per cent. The difference was statistically significant ($p<0.001$). The mean PaO_2 for patients before surgery was 70.933, SD 6.068 mmHg, while the mean PaO_2 for the control was 90.333, SD 6.853 mmHg. The difference was statistically significant ($p<0.0001$). The mean $PaCO_2$ for patients before surgery was 38.567, SD 3.54 mmHg, while the mean $PaCO_2$ for the control was 39.167, SD 1.642 mmHg. The difference was not statistically significant ($p>0.05$).

The comparison between the patients after surgery with the control group regarding the arterial blood gases, showed that the mean O_2 saturation was 93.517, SD 2.993 per cent. The mean O_2 saturation for the control group was 95.917, SD 1.443 per cent. The difference was statistically significant ($p<0.05$). The mean PaO_2 for patients after surgery was 87.833, SD 12.132 mmHg, while the mean PaO_2 for the control was 90.333, SD 6.853 mmHg. The difference was not statistically significant ($p>0.05$). The mean $PaCO_2$ for patients after surgery was 37.6, SD 3.136 mmHg, while the mean $PaCO_2$ for the control was 39.167, SD 1.642 mmHg. The difference was not statistically significant ($p>0.05$).

Discussion

Upper airway obstruction by enlarged adenoids is a frequent problem in children, however, it may be ignored. Trivial complaints such as mouth breathing, snoring, disturbed sleep or frequent chest infections should alert both parents and physician to the serious cardiopulmonary complications which may follow these minor symptoms.

Woodson and Robbins (1985) stated that nasal breathing is of vital importance for both the integrity and function of the lower respiratory tract. In this study, it was found that both the O_2 saturation and PaO_2 were significantly lower in children with enlarged adenoids than the controls. On the other hand, $PaCO_2$ was insignificantly lower in the patients, before adenoidectomy, than the control. Abnormal arterial blood gases mean that uncompensated disease is present, a situation which may be life threatening. It was previously stated that increased airway resistance may be found in children with no overt cardiac symptoms. Despite abnormal pulmonary function and arterial blood gases, these individuals have managed to adjust to their illness. Significantly they are at high risk for sudden cardiac and respiratory failure which may develop under an acute infection of the tonsils and adenoids (Talbot and Robertson, 1973).

The effect of nasal obstruction on pulmonary function can be neural or mechanical (Ogura *et al.*, 1964). It has also been postulated that loss of normal nasal humidification and warming of inspired air could be responsible for changes in diffusion capacity or viscosity of surfactant and may be a potent stimulus for bronchiolar obstruction (Woodson and Robbins, 1985). Edison and Kerth (1973) added that, when the patient becomes a mouth breather due to enlarged adenoids, a part of the stimulus to breathe is lost. This is the force necessary to bring air into the lungs against the nasal resistance.

In this study, as regards arterial blood gases before and after adenoidectomy, there was a statistically significant improvement in the O_2 saturation after surgery ($p<0.0001$). There was also a statistically significant increase in the PaO_2 ($p<0.0001$) and the $PaCO_2$ demonstrated a statistically significant decrease ($p<0.0324$) after the operation. These results coincide with the results published by Macartney *et al.* (1969) and Yonkers and Spaur (1987) after adenotonsillectomy. Macartney *et al.* (1969) and Cayler *et al.* (1969) added that cor pulmonale due to hypoventilation resulting from chronic nasopharyngeal obstruction can lead to gross cardiac failure and yet can apparently be permanently cured simply by removing the tonsils and adenoids.

In conclusion this study showed that the ventilatory impairment associated with enlarged adenoid is reversible after adenoidectomy.

[1] normal value = 95–98%
[2] normal value = 75–100%
[3] normal value = 32–42 mm Hg

References

Belenky, W. (1983) In Pediatric Otolaryngology. First edition. Vol. 1, (Stool Arjona and Bluestone, Eds)., p 712.

Cayler, G. G., Johnson, E. E., Lewis, B. E., Kortzeborn, J. D., Jordan, J., Fricker, G. A. (1969) Heart failure due to enlarged tonsils and adenoids. *American Journal of Diseases of Children,* **118:** 708–717.

Edison, B. D., Kerth, J. D. (1973) Tonsilloadenoid hypertrophy resulting in cor pulmonale. *Archives of Otolaryngology,* **98:** 205–207.

Luke, M. J., Mehrizi, A., Folger, G. M., Rowe, R. D. (1966) Chronic nasopharyngeal obstruction as a cause of cardiopulmonary, cor pulmonale and pulmonary edema. *Pediatrics,* **37:** 762–768.

Macartney, F. J., Pandy, J., Scott, O. (1969) Cor pulmonale as a result of chronic nasopharyngeal obstruction due to hypertrophied tonsils and adenoids. *Archives of Disease in Childhood,* **44:** 585–592.

Menashe, V. D., Farrehi, C., Miller, M. (1965) Hypoventilation and cor pulmonale due to chronic upper airway obstruction. *Pediatrics,* **66:** 198–203.

Ogura, J. H. , Nelson, J. R., Dammkoehler, R., Togawa, K., Kawasaki, M. (1964) Experimental observation of the relation-

ship between upper airway obstruction and pulmonary function. *Annals of Otology, Rhinology and Laryngology,* **73:** 381–403.
Sofer, S., Weinhouse, E., Tal, A., Wanderman, K. L., Margulis, G., Leiberman, A., Gueron, M. (1988) Cor pulmonale due to adenoidal or tonsillar hypertrophy in children. *Chest,* **93:** 119–122.
Talaat, A. M., Nahhas, M. M. (1983) Cardio pulmonary changes secondary to chronic adenotonsillitis. *Archives of Otolaryngology,* **109:** 30–34.
Talbot, A. R., Robertson, L. W. (1973) Cardiac failure with tonsil and adenoid hypertrophy. *Archives of Otolaryngology,* **98:** 277–281.
Woodson, G. R., Robbins, K. T. (1985) Nasal obstruction and pulmonary function. *Otolaryngology—Head and Neck Surgery,* **93:** 505–509.
Yonkers, A. J., Spaur, R. C. (1987) Upper airway obstruction and the pharyngeal lymphoid tissue. *Otolaryngologic Clinics of North America,* **20, 2:** 235–239.

Address for correspondence:
Dr Reda Kamel, M.D.,
5 Dokky Street (Flat 6),
Dokky 12311,
Giza,
Cairo,
Egypt.

Key words: Adenoids; Blood gas monitoring, transcentaneous