EFFECT OF OXYGEN INHALATION ON MICROEMBOLIC SIGNALS IN PATIENTS WITH MECHANICAL AORTIC VALVE

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Abstract- Microembolic signals (MES) are frequently observed in transcranial Doppler (TCD) recordings of patients with mechanical heart valve (MHV). If gaseous bubbles are the underlying cause, number of MES produced by MHV could be reduced with oxygen inhalation. From September 2003 to September 2004, a consecutive series of 14 patients with St Jude aortic valve visited in the cardiology clinic were referred to neurosonology unit, Valie Asr Hospital, Khorasan. TCD monitoring of MES was performed with an ultrasound device and a 2 MHz probe. The MES counts were recorded during 30 minutes breathing room air and thereafter 30 minutes breathing through a facial mask with reservoir bag (6 liter O_2 per minute). The criteria of MES detection were characteristic chirping sound, unidirectional signal, random appearance within cardiac cycle and intensity increase ≥ 3dB above background. The MES counts in two periods of monitoring were compared with paired *t* test and significance was declared at $P < 0.05$. Twelve patients (8 females and 4 males) were investigated. Oxygen ventilation caused a significant decrease of MES counts in the patients in comparison to breathing room air ($P = 0.001$). It seems that MES in patients with MHV are mainly gaseous bubbles caused by blood agitation with MHV. The quantity of MES in patients with MHV is not related to the risk of thromboembolic complications in these patients.

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INTRODUCTION

Thromboembolism is a major cause of morbidity in patients with mechanical heart valve (MHV) and microembolic signals (MES) are commonly observed in transcranial Doppler (TCD) recording of these patients (1). Since the precise nature of MES in patients with MHV is not yet known, different etiologies are under investigation, including local activation of coagulation system by the MHV, local increase of platelet aggregation and gaseous cavitation bubbles (2).

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A major drawback of TCD technology is its failure to provide conclusive information concerning the underlying embolic material (1). This issue is important in evaluation of individual risk profiles and adequate management strategies. No correlations have been found between MES count and duration after MHV replacement, valve position, cardiac rhythm, intensity of anticoagulation and history of neurological deficit (3, 4). Considering gaseous bubbles as embolic material could explain this discrepancy, since microbubbles remain asymptomatic by imploding or crossing over to venous circulation through the capillary bed (4). Patients with MHV have higher MES counts in common carotid artery than middle and anterior cerebral arteries because gaseous bubble emboli are bound to implode with time (5).

This study was carried out to evaluate the influence of oxygen ventilation on MES counts in patients with mechanical aortic valve.

MATERIALS AND METHODS

From September 2003 to September 2004, a consecutive series of patients with St Jude mechanical valve in aortic position were referred from cardiology clinic to neurosonology unit, Vali-e-Asr Hospital, Khorasan.

Presence of more than 70% extracranial internal carotid artery stenosis, poor transtemporal window and intolerance to oxygen ventilation for 30 minutes served as exclusion criteria. The protocol entailed 30 minutes of TCD monitoring while the patient was breathing room air and 30 minutes while breathing 6 liters per minute oxygen through a facial mask with a reservoir bag (fractional inspiratory O_2 = 60%) (6). This facial mask was placed over mouth and nose and held in place by an examiner, providing downward pressure with thumb to ensure a tight seal (6). Patients were instructed to breath normally, avoid hypo or hyperventilation and immediately give notice if breathing became uncomfortable or other inspiratory or cardiac complaints occurred. MES monitoring was performed with an ultrasound device (Vingmed 800, Oslo, Norway) and a 2M Hz probe in 50-58 mm depth of right middle cerebral artery through transtemporal window. MES detection criteria included characteristic chirping sound, unidirectional signal, random appearance in cardiac cycle and intensity increase $\geq$ 3 dB above background. The paired *t* test was applied for comparison of MES counts during oxygen ventilation and resting periods. Significance was declared at $P < 0.05$. The protocol was approved by our institutional ethics committee and the informed consent was obtained from the subjects and/or their guardians.

RESULTS

A total of 14 patients were enrolled in the study. One of these patients did not tolerate oxygen inhalation long enough and developed chest tightness and dizziness that led to immediate termination of oxygen inhalation. The mask could not be tightly applied in an additional patient because of facial hair. Figure 1 shows display of MES in one of the patients. A total of 12 patients (8 females and 4 males) with mean age 38.61 years (SD, 14.41) were investigated. The influence of oxygen ventilation on MES counts in each of the patients is displayed in table 1. We found a significant decrease of MES counts during oxygen ventilation in comparison to resting period ($P = 0.001$).

DISCUSSION

Some basic physiological considerations must be taken into account before analysis of our results. Oxygen inhalation leads to alveolar denitrogenation and at the same time nitrogen washout from the blood. Thus assuming that nitrogen bubbles are underlying embolic material in patients with MHV, one would expect an exponential reduction in MES counts under oxygen inhalation (7). Although all our patients showed significant reduction in MES count during oxygen ventilation, this decreased fraction was not equal between them. Individual differences of denitrogenation procedures depending on lung function could cause this finding. The fraction of inspired oxygen that actively reaches the lung under this procedure is always less than the concentration delivered because of mixing of incoming oxygen with ambient room air entertained by the mask and is strongly dependent on breathing pattern.

Table 1. MES counts of the patients during 30 minutes respiration in room air 30 minutes oxygen ventilation

Patient number	Room air	Oxygen
1	54	30
2	18	1
3	21	6
4	198	30
5	258	48
6	95	30
7	15	2
8	42	12
9	102	4
10	36	21
11	6	0
12	15	0

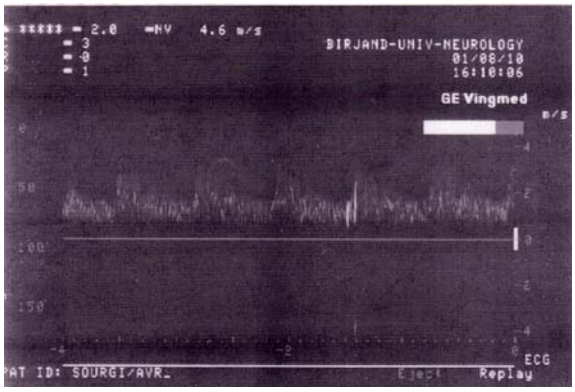

Fig. 1. Display of microembolic signals by transcranial Doppler technology in a patient.

A portion of the detected MES could still arise from coexisting fibrin thrombi on the mechanical aortic valve or native cardiac embolic sources. The quantity of these MES would obviously not be affected by oxygen inhalation (7). Complete elimination of MES during oxygen inhalation was described by Kaps *et al.* (2). It must be stressed that our results are only applicable to patients with MHV and can not be extrapolated to other patients with potential native cardiac or arterial embolic sources. If the underlying embolic material in patients with MHV consisted of cavitation bubbles, no difference in MES counts between neurologically symptomatic and asymptomatic patients would be expected because cavitation bubbles would easily cross over to venous side without causing major vessel obstruction (5). Other studies have shown that MES counts are highest in patients with Bjork-Shiley monostrut valves, significantly lower in St Jude valve carriers and lowest in those with Medtronic-Hall valves (8, 9).

Significant reduction of MES counts in our patients with MHV during oxygen ventilation shows that cavitation bubbles are responsible for a large proportion of the MES in these patients. These cavitation bubbles are probably released during blood agitation through fluid acceleration and deceleration caused by MHV closure. Thus the quantity of MES in patients with MHV is not related to the risk of thromboembolic complications in these patients.

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