Transesophageal echocardiography (TEE) is ideally suited for imaging during cardiopulmonary resuscitation, because high quality images can be obtained without interruption of cardiac compression and ventilation. Such studies have shown that during open-chest cardiopulmonary resuscitation (OCCPR) the cardiac pump mechanism operated with the mitral valve opening during the release phase and closing during the compression phase.1-3 This normal pattern of mitral valve function does not appear to operate in all cases during closed-chest cardiopulmonary resuscitation (CCCPR). In these cases, the mitral valve might remain open during compression of the chest and in the mid-position during the release phase, acting in this way merely as a conduit, while the forward blood flow is generated as the result of the generalized increase in intrathoracic pressure during compression of the chest (thoracic pump). Since the mitral valve seems to be functioning during OCCPR, we sought to
determine during animal experimentation what would be the effect of cardiac massage on a preexisting regurgitant mitral valve.

Methods

Twenty-nine dogs, weighing 18-30 kg, were used as experimental animals. The study was performed in accordance with the ethical standards established by IASP Guiding Principles for Research Involving Animals and Human Beings, concerning the care and use of animals. After anesthesia with intravenous administration of sodium thiopental (Pentothal), tracheal intubation was performed and ventilation was secured using a Harvard respirator. A transverse thoracotomy was then performed, the pericardium was opened and a 5.0 MHz monoplane transesophageal echocardiographic probe was positioned in the lower esophagus to obtain the two-dimensional transverse four-chamber view. Attempts to produce acute mitral regurgitation (aMR) were made using a specially designed metal probe with a hook-like upper end that was introduced to the left ventricular cavity through the apex (Figure 1). The attempts to induce mitral apparatus damage were directed mainly towards the chordae tendinae and secondarily towards the papillary muscles or commissures. The aforementioned procedure was performed under online transesophageal echocardiographic guidance. The hole in the left ventricular apex caused by the metal probe was sutured before any resuscitation attempt. Thirteen of the animals showed ventricular fibrillation or electromechanical dissociation during the attempts. Open-chest cardiopulmonary resuscitation was initiated immediately to restore the cardiac function. The massage was performed with the one-hand technique. The heart was placed in the palm of the right hand and squeezed between the palm and the thumb in a milking fashion from the apex to the base of the heart. With the TEE on line, the cardiac structure and function were monitored during OCCPR and recorded. The regurgitant area of the mitral jet in cm² was calculated before the attempt to produce aMR and then during OCCPR. The left ventricular (LV) transverse end-systolic diameter in cm and the volume in cm³ were also calculated before and during OCCPR. At the end of the experiments autopsy of all the hearts was performed. The paired Student’s t-test was used for statistical evaluation.

Results

In 20 of the 29 dogs (69%) there was a mild degree of central mitral regurgitation before any attempts were made to produce aMR. Ventricular fibrillation or/and electromechanical dissociation occurred in 13 animals (13/29, 45%). In 9 of these 13 dogs there was no severance of the mitral apparatus found at autopsy, despite the attempts to produce aMR. In 5 of these 9 there was mild preexisting mitral regurgitation, while in the remaining 4 no mitral regurgitation was detected. In the other 4 dogs of the 13, there was severance of the mitral apparatus at autopsy and mitral regurgitation was found by echocardiography.

During OCCPR in the 4 animals without preexisting mitral regurgitation, there was no mitral regurgitation detected by TEE (Figures 2 & 3), while in the oth-
er 5 with preexisting mitral regurgitation a slight increase in the degree of the regurgitant flow was noticed. The regurgitant flow area changed from 0.62 ± 0.17 cm² before to 1.1 ± 0.36 cm² during OCCPR (p<0.008, Table 1). In the 4 animals with severance of the mitral apparatus after the attempts (Figures 4 & 5) and prolapse of the cusps within the left atrium, the regurgitant flow increased from 4.01 ± 0.93 cm² before to 7.7 ± 2.6 cm² during OCCPR (p<0.002, Table 2). The LV transverse diameter decreased from 4.39 ± 0.62 cm before to 1.54 ± 0.5 cm during OCCPR (p<0.001) and the LV volume from 60.5 ± 7.14 cm³ to 17.02 ± 4.14 cm³ (p<0.001), thus showing the effectiveness of OCCPR.

Discussion

The first apparently successful open-chest cardiac massage was reported in animals in the second half of the nineteenth century. Although later, in the middle of the 20th century, Kouwenhoven and his colleagues popularized CCCPR, there are still indications nowadays for performing OCCPR in certain cases, with improved survival. The open-chest cardiac massage (OCCM) is more efficient than closed chest compressions in cardiac arrest, having been proved to produce better coronary perfusion coronary pressure than standard CPR. However, OCCM is used less often than closed-chest cardiac compressions, because the most limiting factor in this technique is the necessity to perform a thoracotomy in order to insert the hand and compress the heart. OCCM may be indicated for patients with cardiac arrest due to trauma, or in the early postoperative phase after cardiothoracic surgery.

Echocardiographic studies have demonstrated that during OCCPR the mitral valve closes during the compression phase of the cardiac massage and opens during the release phase (cardiac pump mechanism). When CCCPR was first applied, it was thought that the forward blood flow was produced by compression of the heart between the sternum and the spine. Although this could happen, another mechanism also seems to be in operation. This so-called thoracic pump mechanism refers mainly to the differential transmission during chest compression of the increased intrathoracic pressure to extrathoracic arteries and veins, creating a pressure difference and therefore a forward blood flow. The heart serves simply as a conduit, with the mitral valve staying open. The function of the mitral valve therefore remains the key feature, whichever mechanism, either the cardiac pump or the thoracic pump model, operates directly or in combination to produce forward blood flow. In this study we sought to determine what would be the function of the mitral valve when there was pre-existing regurgitation during OCCPR and the forward blood flow was produced solely by the mechanism of

Table 1. Mitral regurgitation jet area (cm²) before and during cardiac massage (CM), in five animals with preexisting mitral regurgitation.

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<td>Before CM</td>
<td>0.6</td>
<td>0.7</td>
<td>0.55</td>
<td>0.4</td>
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<td>During CM</td>
<td>1.1</td>
<td>1.0</td>
<td>0.95</td>
<td>0.75</td>
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the cardiac pump. Our data indicate that, if the preexisting mitral regurgitation is chronic and of mild degree, there is only a small increase in the regurgitant flow, whereas when the pre-existing mitral regurgitation is acute and of moderate degree, the regurgitant flow almost doubles. A preexisting competent mitral valve, however, remains competent during the cardiac massage.

**Conclusions**

The results of our study may have an important clinical implication. Given the presence of preexisting mitral regurgitation, direct cardiac massage during resuscitation efforts would probably be less effective than expected, because of an increase in the amount of the regurgitant flow, and the practicing physician should be aware of this possibility. Caution, however, should be exercised when our data are applied to CCCPR. In this case, the heart pump mechanism could operate alone or in combination with the thoracic pump mechanism and the effect of preexisting mitral regurgitation might be different.

**Table 2.** Mitral regurgitation jet area (cm²) before and during cardiac massage (CM), in four animals with severance of the mitral apparatus due to chordae tendineae damage.

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<tr>
<td>Before CM</td>
<td>2.8</td>
<td>3.8</td>
<td>4.9</td>
<td>4.55</td>
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<tr>
<td>During CM</td>
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**References**