Variations in the papillary muscles of normal tricuspid valve and their clinical relevance in medicolegal autopsies

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ABSTRACT

Objective: In our study, tricuspid valves in cases of sudden death secondary to congenital differences of the tricuspid valve with significant papillary muscle anatomy were investigated. No studies of papillary muscle anatomy of the tricuspid valve have been found in medicolegal autopsies in literature. The purpose of our study is to investigate the relationship of papillary muscle in tricuspid valve in cases of sudden deaths, especially those resulting from cardiac disease, with the muscle structure, as well as the number of the muscle leading congenital changes.

Methods: The study was carried out in the Department of Anatomy, Faculty of Medicine, Ege, University, Izmir, Turkey and comprised of 400 human hearts obtained between 2000 and 2002 from 400 autopsy cases during a medicolegal autopsy with permission from the Council of Forensic Medicine, Izmir. Quantitative and morphological aspects of the papillary muscles of the right ventricle were evaluated. The criteria such as number, incidence, length and shape of the anterior, (APM) septal (SPM) and posterior papillary muscles (PPM) have been observed.

Results: Although the papillary muscle presented great variability in numbers, with a minimum of 2 and a maximum of 9 papillary muscles in the right ventricle, there were usually 3 papillary muscles in the right ventricle; APM, PPM and SPM. The one headed APM was found to be more often in cardiac deaths. However, observing more frequent conical and flat topped configurations in all PPM was striking. The absence or lower ratio, or both of attachment bridges of SPM and APM/PPM in deaths of cardiac origin is also significant. We have found that the presence of this attachment is higher in deaths of noncardiac origin.

Conclusion: This anatomical study may explain the increased in incidence wide variations of papillary muscle tricuspid valve in deaths of cardiac origin. The verdict in legal affairs may change with this. The knowledge regarding wide variations and minor anatomical abnormalities of papillary muscle helps forensic examiners not to get confused at unexpected deaths.

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The papillary muscles play an important role in right ventricular contraction by drawing tricuspid annulus toward the apex, thereby causing shortening of the long axis and sphericity of the chamber for ejecting blood.[1,2] The base for the description of the papillary muscle of the right ventricle and its anatomic variations were reported by Silver et al[3] Acar et al[4] and Wafae et al[5] all elegantly described the anatomy of the tricuspid valve. The relevance of chordopapillary variations in rheumatic heart disease, reparative procedures, papillary muscle dysfunction, mitral valve prolapse, mitral valve replacement and use of mitral valve homograft for mitral/tricuspid replacement have

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been discussed in previous studies.[6-8] The left atrioventricular valve and its papillary muscle and chordae tendineae have been largely studied.[9-13] In particular, the observation of papillary muscle and chordae tendineae of the right ventricle have shown endless variability in their number, shape and location.[14,15] The number, size, location and morphology of the right papillary muscle and their correlation to age and race were studied in an attempt to provide a classification.[16] Congenital variations are known to be potential candidates for mechanical trauma leading to tricuspid valve lesions.[17,18] Minor degrees of variations have no more effect than changing heart function.[8,16] A damage to valves or papillary muscles may occur after a trauma and may further even lead to death, negatively affecting valve functions and creating losses in the functional capacity of the body.[19] Controversies on the importance of these variations still exist and put the human beings at risk of unexpected deaths.[20] Over time, progressive regurgitation can lead to right ventricular failure and arrhythmias, and death in such people.[21] Whether a relative connection between death and trauma which is not the direct cause of death exists concerns the researchers. For this reason, a detailed examination of heart valves as well as papillary muscles should carefully be performed to clarify the reason for sudden death with no apparent cause. That the lack of papillary muscle in mitral valve leads to congenital mitral regurgitation is known.[1] We studied whether this is also true in the cases of tricuspid valve. Increasing clinical and surgical importance of the papillary muscle and lack of available detailed information on the anatomical characteristics of the in sudden deaths have led us to carry out this investigation. The aim of this study is to investigate the relationship of the papillary muscle in the tricuspid valve in cases of sudden deaths, especially those caused by cardiac diseases with muscle the structure, as well as the number of the muscles leading to congenital changes.

Methods. The materials were collected from 400 death cases (200 of cardiac origin and 200 of noncardiac origin) during a medicolegal autopsy with the permission of the Council of Forensic Medicine, Ministry of Justice, Izmir, Turkey. We studied hearts with no macroscopic evidence of rheumatic and congenital lesions. There were 2 groups, both from cardiac and noncardiac origins of death including 60 hearts from females and 140 hearts from males. The age of the individuals, whose autopsies were performed between 2000-2002 in Izmir, varied from 17-68-years. In all cases, a complete autopsy, toxicology screening and histological examinations were performed. Deaths of unnatural cause or from alcohol and overdose of drugs were excluded from the study. Each heart was sectioned along its acute margin. The section passed near the anteroposterior commissure of the right atrioventricular valve, with an incision from the right atrium to the apex of the right ventricle. After opening, each heart was washed under tap water to remove blood clots. Dissection of the myocardium was carried out from the right atrioventricular fibrous ring to the origin of papillary muscles, preserving the integrity of the valve apparatus as a whole. We studied the morphometric and morphologic data obtained from each papillary muscle: the width and length of the papillary muscle, the number of the papillary muscle in association with the presence of the septal papillary muscle (SPM), and their relation with the leaflets. Data were recorded by statistical package for social sciences for Windows.

Results. The mean age of the cases was 40.9. In the group with cardiac origin of death (group 1), the most common single cause was ischemic cardiovascular disease, which accounted for 158 (79.2%) of the deaths due to cardiovascular diseases. In the one with noncardiac origin of death (group 2), respiratory and central nervous system diseases ranked as the first (45%) and second (32%) as regards incidence. Other origins, such as malignant, hepatic, renal, gastrointestinal, endocrinal, septicemic, epileptic, and aortic aneurysmatic, had relatively little significance.

Papillary muscle. The papillary muscles were conical projections of the ventricular muscle the apices which afforded attachment to the chordae tendineae **(Figures 1 & 2)**. Although the papillary muscle presented great variability in number, with a minimum of 2 and a maximum of 9 papillary muscle in the right ventricle **(Figure 3)**, there were usually 3 papillary muscles (anterior, (APM) posterior (PPM) and SPM in the right ventricle that corresponded nominally to the leaflets of the tricuspid valve. The single papillary muscles were conical **(Figure 3)**, mamillated **(Figure 4)**, flat topped **(Figure 2)**, grooved **(Figure 5)**, stepped **(Figure 6)**, wavy, arched, sloped or saucerized. When there were 2 bellies, they presented a 2 tiered, interlinked, parallel **(Figure 7)**, arched, V, Y or H configuration **(Figure 8)**. Papillary muscles with 3 bellies formed a parallel, interlinked or arched arrangement **(Figure 9)**. When 4 or 5 bellies existed; they were parallel or interlinked. The muscles that originate from the ventricular wall of the right ventricle were longer and larger than those arising from the septal wall. All the major papillary muscles supply chordae to adjacent components of the leaflets they support.

The connections of the SPM with anterior and PPM were observed **(Figures 4, 8 & 10)**. The attachments of papillary muscles to the ventricular

wall were thin and long. These attachments were 4.5-15 mm in length and 9-20 mm in thickness. The distribution and incidence of the APM, PPM and SPM have been shown in **Tables 1, 2 & 3**. We identified hematoma in the structure of papillary muscles of 20 individuals **(Figure 1)**. In 3 cases were found rupture of papillary muscles **(Figure 2)**. We concluded that rupture was caused by increased tension in the ischemic papillary muscle related to preservation of the chordae. The papillary muscle showed evidence of ischemia when examined histologically. We decided hemorrhagic necrosis of the papillary muscle, possibly related to subendocardial ischemia, to be the cause of rupture in one case.

Anterior papillary muscle. In noncardiac death cases. The APM was present in 100% of the hearts: it was the largest papillary muscle, and the most prominent of the 3 **(Figure 10)**. The APM was often bifid, owing to 2 papillae arising from its apex; 3 or 4 papillae were rarely found. The APM was found protruding partly from the anterior and partly from the septal wall of the right ventricle. The papillae always formed the origins of the chordae tendineae. Its chordae tendineae were attached to the anterior and posterior leaflets of the tricuspid valve, and frequently a portion of that muscle constituted the moderator band **(Figure 11)**. When 2 papillary muscles were present, the smaller muscle gave origin to a lower number of chordae tendineae.

We found a single APM in 80.5% of the cases (161 cases) and a double one in 19.5% (39 cases). We observed the double APM to be more common in males than in females. A double APM was found associated with the lack of SPM in 4 cases (2.5%) and with the presence of a muscular bridge connecting the two heads. The most common shapes of APM were conical and flat-topped, according to Victor's classification. It was found that more complex configurations, such as V, Y, H and parallel like, were more prominent in deaths of noncardiac origin **(Table 4)**. The APM bellies were mostly intraluminal in 15%, mostly intraluminal with the tip anchored in 20%, equally sessile and intraluminal in 52%, mostly sessile in 13%. In this group, 17% of the APM bellies arose from the upper third of the ventricle, 80% from the middle third, and 3% from the lower third. Anterior papillary muscle was found to have the major length. In many cases, one large and one long APM have been found **(Figure 11).** A single APM was found with a length ranging between 11.45 and 45.3 mm (mean 20), and this was greater than that of a double one ranging between 7.2 and 25.15 mm (mean 15) **(Table 5)**.

Association with the presence of a septal papillary muscle. The one headed APM (n:161) was associated with the presence of at least one SPM in 144 hearts and 89.4% of the cases were distributed as follows: 81 hearts presented one SPM (50.3%), 28 presented 2 SPM (17.4%), 21 presented 3 SPM (13%) and 14 presented 4 SPM (8.7%) **(Figure 8)**. The one headed APM was not associated with the presence of the SPM in 17 hearts, namely 10.6% of the cases **(Table 6)**. The double headed APM (n: 39) was associated with the presence of at least one SPM in 32 hearts, 82.1% and were distributed as follows: 12 hearts presented one SPM (30.8%), 9 presented 2 SPM (23.1%), 6 presented 3 SPM (15.4%) and 5 presented 4 SPM (12.8%). The double headed APM was not associated with the existence of the SPM in 7 hearts (17.9%).

In cardiac death cases. The APM was present in 99.5% of the hearts **(Table 1).** The APM was usually conical and flat topped, owing to 2 papillae arising from its apex; 3 or 4 papillae were rarely found **(Figure 2)**. We found a single APM in 83.5% of the cases (167 cases), double ones in 13.5% (27 cases) and nil in 3% (6 cases). We observed the double APM to be more common in males than in females. The APM bellies were mostly intraluminal, intraluminal with the tip anchored, equally sessile and intraluminal and mostly sessile, 18%, 20%, 50% and 12%. In group one, 15% of the APM bellies arose from the upper third of the ventricle, 80% from the middle third, and 5% from the lower third. The length of the APM ranged from 6.2-52.1 mm (mean 19.16). The length of a single-headed APM ranged from 10.9-52.1 mm (mean 21). The range was between 6.2 and 24.12 mm (mean 17) for a double headed APM.

Association with the presence of a septal papillary muscle. The one headed APM (n:167) was associated with the presence of at least one SPM in 100 hearts, 60%, and the cases were distributed as follows: 34 hearts presented one SPM (20.4%), 28 presented 2 SPM (16.8%), 22 presented 3 SPM (13.2%) and 16 presented 4 SPM (9.6%). The one-headed APM was not associated with the presence of the SPM in 67 heart, namely 40% of the cases **(Table 7)**. The double headed APM (n=27) was associated with the presence of at least one SPM in 15 hearts, 55.5%, and were distributed as follows: 7 hearts presented one SPM (25.9%), 5 presented 2 SPM (18.5%), 2 presented 3 SPM (7.4%) and one presented 4 SPM (3.7%). The double headed APM was not associated with the existing SPM in 12 hearts (44.5%).

Posterior papillary muscle. Noncardiac death cases. The PPM was present in 100% of the hearts. One and 2 PPM were frequently observed (26.5% and 48.5%). Three and 4 PPM were found in 34 and 15 cases (17% and 7.5%). Five PPM was found in one case (0.5%) **(Table 2)**. In the PPM, the papillary muscle bellies were mostly intraluminal in 14%, mostly intraluminal with the tip anchored in 19%, equally sessile and intraluminal in 54.5%, mostly sessile in 12.5%. In group one, 24% of papillary muscle bellies arose from the upper third

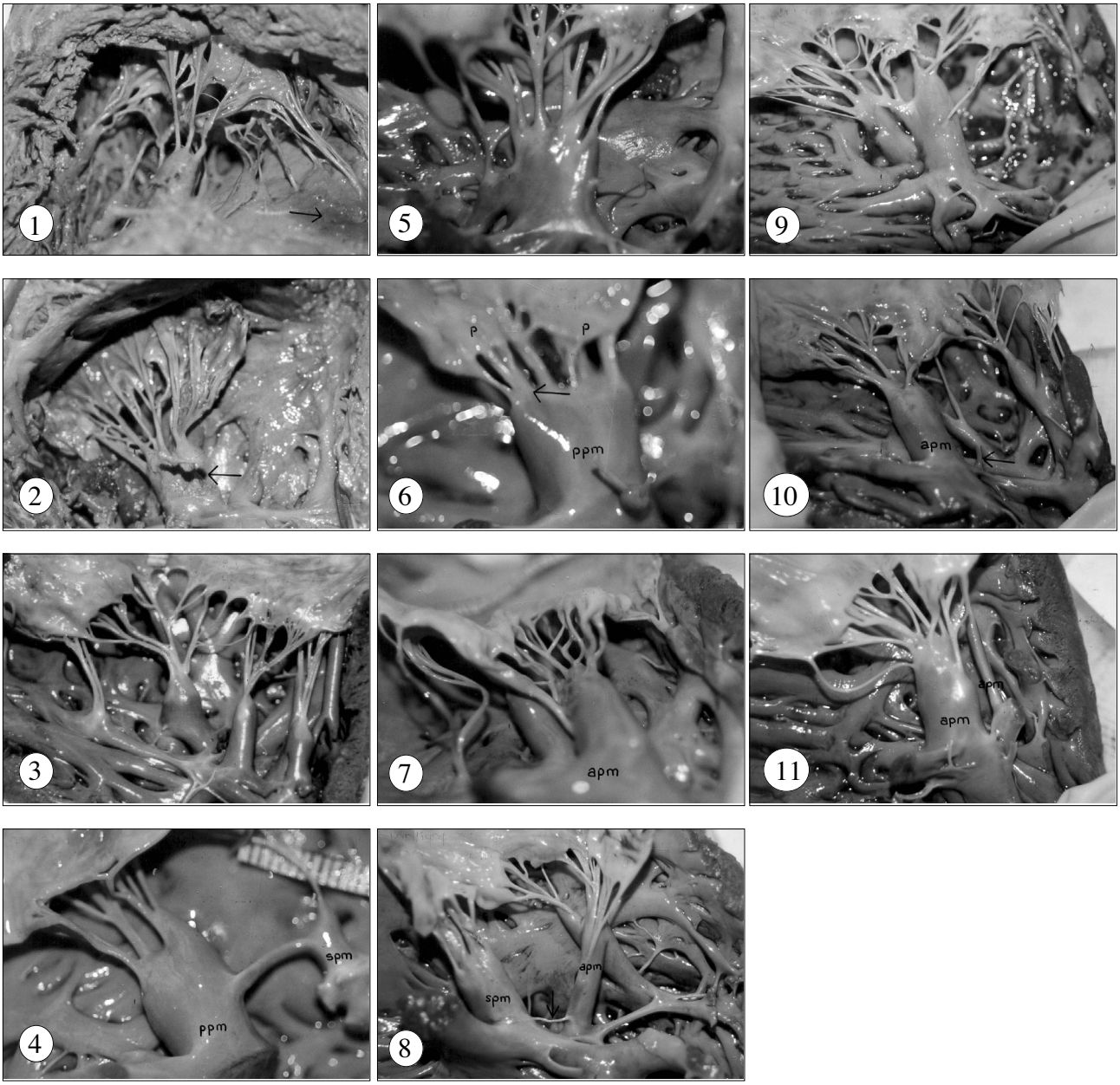

Figure 1 - Papillary muscles were conical projections of the ventricular muscle. Hematoma was the identified structure of the papillary muscle (arrow).

Figure 2 - Flat topped type papillary muscle was ruptured in cardiac death cases.

Figure 3 - Four papillary muscles were not connected by a muscular bridge at their origin.

Figure 4 - The septal papillary muscle (spm) was rudimental, and the posterior papillary muscle (ppm) was mamilled.

Figure 5 - Single anterior papillary muscle had grooved configuration.

Figure 6 - Single posterior papillary muscle (ppm) was step-shaped.

Figure 7 - Parallel configuration of the anterior papillary muscle (apm) and no septal papillary muscles.

Figure 8 - X configuration of the anterior papillary muscle (apm) which was connected to the septal papillary muscle (spm) by a muscular bridge (arrow).

Figure 9 - Three-bellied papillary muscle formed an interlinked configuration.

Figure 10 - The anterior papillary muscle (apm) was the largest papillary, with the presence of a muscular bridge (arrow) of the septal papillary muscle.

Figure 11 - The one large and one long anterior muscle have been shown. Anterior papillary muscle (apm)

Table 1 - Distribution of the anterior papillary muscle in cardiac and noncardiac death cases.

Papillary muscle heads	Males				Females				Total			
	Noncardiac		Cardiac		Noncardiac		Cardiac		Noncardiac		Cardiac	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
1	105	(75)	110	(78.6)	56	(94)	57	(95)	**161**	**(80.5)**	**167**	**(83.5)**
2	35	(25)	25	(17.9)	4	(6)	2	(3.3)	**39**	**(19.5)**	**27**	**(13.5)**
Absent	-		5	(3.5)	-		1	(1.7)	**-**		**6**	**(3)**

Table 2 - Distribution of the posterior papillary muscle in cardiac and noncardiac death cases.

Papillary muscle heads	Males				Females				Total			
	Noncardiac		Cardiac		Noncardiac		Cardiac		Noncardiac		Cardiac	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
1	38	(27)	50	(35.7)	15	(25)	36	(60)	**53**	**(26.5)**	**86**	**(43)**
2	70	(50)	65	(46.5)	27	(45)	11	(18.3)	**97**	**(48.5)**	**76**	**(38)**
3	29	(21)	14	(10)	5	(8.3)	2	(3.3)	**34**	**(17)**	**16**	**(8)**
4	3	(2)	8	(5.7)	12	(20)	7	(11.7)	**15**	**(7.5)**	**15**	**(7.5)**
5	-		1	(1.7)	1	(1.7)	1	(1.7)	**1**	**(0.5)**	**2**	**(1)**
Absent	-		2	(1.4)	-		3	(5)	**-**		**5**	**(2.5)**

Table 3 - Distribution of the septal papillary muscle in cardiac and noncardiac death cases.

Papillary muscle heads	Males				Females				Total			
	Noncardiac		Cardiac		Noncardiac		Cardiac		Noncardiac		Cardiac	
	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)	n	(%)
1	28	(20)	30	(21.4)	15	(25)	10	(16.7)	**43**	**(21.5)**	**40**	**(20)**
2	56	(40)	55	(39.3)	24	(40)	25	(41.6)	**80**	**(40)**	**80**	**(40)**
3	28	(20)	36	(25.7)	6	(10)	10	(16.7)	**34**	**(17)**	**46**	**(23)**
4	3	(2)	12	(8.6)	10	(17)	5	(8.3)	**13**	**(6.5)**	**17**	**(8.5)**
Absent	25	(18)	7	(5)	5	(8)	10	(16.7)	**30**	**(15)**	**17**	**(8.5)**

Table 4 - Distribution of different types of right-sided papillary muscles (pm) in cardiac and noncardiac (noncardi) death cases.

PM	Shape	Conical	Flat topped	Mamilled	Arched	Wavy	Grooved	Stepped	Sloped	Saucerized	V-Y-H form	Parallel	Tiered
apm	Cardiac	21%	20.5%	17%	3.2%	2.1%	2.2%	3%	7.4%	2.6%	5%	6%	10%
	Noncardi	10.4%	18.5%	10.2%	2.4%	1.2%	2.5%	4.2%	3%	6.1%	20.5%	18.2%	2.8%
ppm	Cardiac	25.2%	17.7%	18.2%	2.7%	3.1%	2%	2.9%	6.8%	1.9%	3.5%	7.5%	8.5%
	Noncardi	11.2%	17.1%	15.4%	1.2%	1.7%	2.1%	2.3%	2.7%	2.9%	21.7%	18.3%	3.4%
spm	Cardiac	30.7%	16.3%	18.5%	3.2%	3.9%	1.9%	3.2%	7.2%	1%	4.2%	6.3%	3.2%
	Noncardi	10.9%	17.4%	9.2%	1.8%	1.7%	2.6%	3.1%	2.9%	5.7%	22.1%	17.3%	5.3%

Table 5 - Length of the anterior, posterior and septal papillary muscle in cardiac and noncardiac death cases.

Muscle	Heads	Noncardiac death min-max (mean) size (mm)	Cardiac death min-max (mean) size (mm)
APM	1 head	11.45-45.3 (20)	10.9-52.1 (21)
	2 heads	7.2-25.15 (15)	6.2-24.12 (17)
PPM	1 head	2-31 (15)	3-33 (16.9)
	2 heads	4.4-16.24 (8.8)	4.2-17.15 (9.2)
	3 heads	2.8-15.2 (7.47)	2.6-16.2 (7.5)
	4 heads	3.7-8.15 (6.48)	3.5-7.95 (7.8)
	5 heads	5.27	2-6.42 (6.4)
SPM	1 head	3-15.04 (5.6)	2.7-15.75 (6.2)
	2 head	1.28-12 (4.5)	1.3-12.25 (5.2)
	3 head	1.47-14 (4.5)	1.45-13.75 (5.2)
	4 head	2.67-5 (2.81)	2.5-7 (3.4)

APM - anterior papillary muscle, PPM - posterior papillary muscle
SPM - septal papillary muscle

Table 6 - Association of the anterior and posterio papillary muscles with the presence of the septal papillary muscle in noncardiac death cases.

SPM	Heads of APM 1 n	(%)	2 n	(%)	Heads of APM 1 n	(%)	2 n	(%)	3 n	(%)	4 n	(%)
1	81	(50.3)	12	(30.8)	22	(41.5)	32	(33)	12	(35.3)	5	(33.4)
2	28	(17.4)	9	(23.1)	12	(22.6)	25	(25.8)	8	(23.5)	3	(20)
3	21	(13)	6	(15.4)	7	(13.2)	17	(17.5)	6	(17.6)	3	(20)
4	14	(8.7)	5	(12.8)	5	(12.8)	6	(6.2)	4	(11.8)	2	(13.3)
None	17	(10.6)	7	(17.9)	7	(17.9)	17	(17.5)	4	(11.8)	2	(13.3)

APM - anterior papillary muscle, SPM - septal papillary muscle

Table 7 - Association of the anterior and posterior papillary muscles with the presence of the septal papillary muscle in cardiac death cases.

SPM	Heads of APM 1 n	(%)	2 n	(%)	Heads of APM 1 n	(%)	2 n	(%)	3 n	(%)	4 n	(%)
1	30	(20.4)	7	(25.9)	13	(15.1)	21	(27.6)	4	(25)	2	(13.3)
2	28	(16.8)	5	(18.5)	10	(11.6)	18	(23.7)	2	(12.5)	2	(13.3)
3	22	(13.2)	2	(7.4)	9	(10.5)	11	(14.4)	1	(6.3)	1	(6.7)
4	16	(9.6)	1	(3.7)	8	(9.3)	5	(6.6)	1	(6.3)	1	(6.7)
None	67	(40)	12	(45.5)	46	(53.5)	21	(27.6)	8	(50)	9	(50)

All data were significant ($p<0.05$)
APM - anterior papillary muscle, SPM - septal papillary muscle

of the ventricle, 75% from the middle third, and 1% from the lower third. The length of the PPM ranged from 2-31 mm (mean 11), while that of a single headed APM ranged between 2 and 31 mm (mean 15). The length of a double headed PPM ranged from 4.4 and 16.2 mm (mean 8.8), whereas ranged was between 2.8 and 15.2 mm (mean 7.47) for a 3-headed PPM. The length of a 4 headed PPM ranged from 3.7-8.15 mm (mean 6.48) and it was 5.27 mm for a 5 headed PPM **(Table 5)**.

Association with the presence of a septal papillary muscle. The one headed PPM (n=53) was associated with the presence of at least one SPM in 48 hearts, 90.5%, and the cases were distributed as follows: 22 hearts presented one SPM (41.5%), 12 presented 2 SPM (22.6%), 7 presented 3 SPM (13.2%) and 7 presented 4 SPM (13.2%). The PPM was not associated with the presence of the SPM in 5 that is 9.5% of the cases **(Table 6)**. The double headed PPM (n:97) was associated with the

presence of at least one SPM in 80 hearts, 82.5% and these were distributed as follows: 32 hearts presented one SPM (33%), 25 presented 2 SPM (25.8%), 17 presented 3 SPM (17.5%) and 6 presented 4 SPM (6.2%). The double headed PPM was not associated with the existence of the SPM in 17 hearts (17.5%). A 3 headed PPM (n=34) was associated with the presence of at least one SPM in 30 cases, 88.2% and these were distributed as follows: 12 hearts presented one SPM (35.3%), 8 presented 2 SPM (23.5%), 6 presented 3 SPM (17.6%) and 4 presented 4 SPM (11.8%). The 3-headed PPM was not associated with the SPM's presence in 4 hearts (11.8%). A 4 headed PPM (n=15) was associated with presence of at least one SPM in 13 cases, (86.7%), and were distributed as follows: 5 hearts presented one SPM (33.4%), 3 presented 2 SPM (20%), 3 presented 3 SPM (20%) and 2 presented 4 SPM (13.3%). The 4 headed PPM was not associated with presence of the SPM in 2 hearts (13.3%) **(Figure 3)**.

In cardiac death cases. The PPM was present in 97.5% of the hearts. The number and morphology of the PPM were highly variable and smaller than those of the APM. The PPM consisted of 2 or 3 parts which arose from the inferior wall of the right ventricle, and to which chordae tendineae of the posterior and septal leaflets were attached **(Figure 6)**. One and 2 PPM were frequently observed (43% and 38%). Three and 4 PPM were found in 16 and 15 cases (8% and 7.5%). Five PPM was found in 2 cases (1%). The PPM was not present in 5 cases (2.5%) **(Table 2)**. In the PPM, the papillary muscle bellies were mostly intraluminal in 14%, mostly intraluminal with the tip anchored in 19%, equally sessile and intraluminal in 54.5%, mostly sessile in 12.5%. In this group, 24% of the papillary muscle bellies arose from the upper third of the ventricle, 75% from the middle third, and 1% from the lower third. When 2 papillary muscles were present, the smaller muscle gave origin to a lower number of chordae tendineae. The length of the PPM ranged from 3-33 mm (mean 11). The length of a single headed APM ranged from 3-33 mm (mean 16.9), while that of a double headed PPM ranged from 4.2 and 17.15 mm (mean 9.2). The length of a 3 headed PPM ranged from 2.6-16.2 mm (mean 7.7), whereas the range was between 3.5 mm and 7.95 mm (mean 7.8) for a 4 headed PPM. The length of a 5 headed PPM ranged from 2-6.42 mm (mean 6.4) **(Table 5)**.

Association with the presence of a septal papillary muscle. The one headed PPM (n:86) was associated with the presence of at least one SPM in 40 hearts, 46.5%, and the cases were distributed as follows: 13 hearts presented one SPM (15.1%), 10 presented 2 SPM (11.6%), 9 presented 3 SPM (10.5%) and 8 presented 4 SPM (9.3%). The one headed PPM was not associated with the presence of the SPM in 46 hearts that is, 53.5% of the cases **(Table 7)**. The double headed PPM (n=76) was associated with the presence of at least one SPM in 55 hearts, 72.4%, and were distributed as follows: 21 hearts presented one SPM (27.6%), 18 presented 2 SPM (23.7%), 11 presented 3 SPM (14.4%) and 5 presented 4 SPM (6.5%). The double headed PPM was not associated with the SPM's presence in 21 hearts (27.6%). A 3 headed PPM (n=16) was associated with the presence of at least one SPM in 8 cases, 50%, and were distributed as follows: 4 hearts presented one SPM (25%), 2 presented 2 SPM (12.5%), one presented 3 SPM (6.3%) and one presented 4 SPM (6.3%). The 3-headed PPM was not related with the presence of the SPM in 8 hearts (50%). A 4 headed PPM (n:15) was associated with the existence of at least one SPM in 6 cases that is, 40%, and were distributed as follows: 2 hearts presented one SPM (13.3%), 2 presented 2 SPM (13.3%), one presented 3 SPM (6.7%) and one presented 4 SPM (6.7%). In 9 hearts (60%), no single SPM was found when a 4-headed PPM was present.

Septal papillary muscle in noncardiac death cases. The SPM was present in 170 hearts (85%). The SPM was rudimentary, often absent or very small in size **(Figure 4)**. The SPM presented one, 2, 3 or 4 heads. The incidence of the double SPM (40%) was more frequent than that of the single one (21.5%). Three or 4 SPM were found in 17% and 6.5% of the cases. In 30 cases (15%), no SPM was found **(Table 3)**. In these cases, the chordae tendineae were directly attached to the ventricular wall **(Figures 3 & 7)**. When one SPM existed, it presented a small head with only one tip forming chordeae tendineae heading to septal and anterior leaflets. When more then one SPM was observed, each muscle had a very small head, not comparable in size with APM and PPM; the group of more than one SPM has been defined as medial papillary complex. The SPM bellies were mostly intraluminal in 13%, mostly intraluminal with the tip anchored in 18%, equally sessile and intraluminal in 58% and mostly sessile in 11% of the cases. In this group 17% of the papillary muscle bellies arose from the upper third of the ventricle, 80% from the middle third, and 3% from the lower third. Its length ranged from 1.28-15.04 mm (mean 5.02). The length of a single headed SPM ranged from 3-15.04 mm (mean 5.6), while that of a double headed SPM ranged from 1.28-12.02 mm (mean 5.6). The length of a 3 headed SPM ranged from 1.47-14 mm (mean 4.50), whereas the range was 6.67-5 mm (mean 2.81) for a 4 headed SPM **(Table 5)**.

In cardiac death cases. The SPM was present in 183 hearts (91.5). The incidence of the double SPM (20%) was more frequent than that of the single one (40%). Three or 4 SPM were found in 23% and 8.5% of the cases. In 17 cases (8.5%), no SPM was found **(Table 3)**. The length ranged from

1.3-15.75 mm (mean 5.04). The length of a single headed SPM ranged from 2.7-15.75 mm (mean 6.2), while that of a double headed SPM ranged from 1.3-12.25 mm (mean 5.2). The length of a double-headed SPM ranged from 1.28-12.02 mm (mean 5.6), whereas the range varied between 1.45-14 mm (mean 5.20) for a 3 headed SPM. The length of a 4 headed SPM ranged from 2.5-7 mm (mean 3.4) (**Table 5**). The SPM bellies were mostly intraluminal in 14%, mostly intraluminal with the tip anchored in 18%, equally sessile and intraluminal in 58% and mostly sessile in 10%. In this group, 16% of the papillary muscle bellies arose from the upper third of the ventricle, 81% from the middle third, and 3% from the lower third.

Discussion. There is limited research on physical, ontogenetic and forensic studies of the papillary muscles of tricuspid valve anatomy in sudden death.

The results of this anatomical study may explain the increased incidence in wide variations of papillary muscle tricuspid valve in deaths of cardiac origin. Short, thick or V, Y and X configuration of papillary muscle tendineae differing from normal can be easily ruptured with chest trauma. This may exhibit individual differences. In the hearts investigated in previous studies, no examination based on the cause of death has been carried out. The present study is significant in that it has been based on the origin of death. In this study, the one headed APM was found to be more often in cardiac death cases. However, observing more frequent conical and flat topped configurations in all PPMs was striking. The absence or lower ratio or both of the attachment bridges of SPM and APM/PPM in deaths of cardiac origin was also significant. We found that the presence of this attachment was higher in noncardiac originated deaths. We think that this condition negatively affects the feeding of papillaries and acts as a culprit of cardiac deaths, for aging starts at an early stage. On the other hand, the papillary muscle showed evidence of ischemia when examined histologically. We decided hemorrhagic necrosis of the papillary muscle, possibly related to subendocardial ischemia, to be the cause of rupture in one case. We concluded that rupture was caused by increased tension in the ischemic papillary muscle related to preservation of the chordae. For this reason, we recommend that attempts be made to avoid tension on the papillary muscle whenever chordal preservation is performed, particularly when the chordal attachments to the valve leaflets are altered. As a result of studying literature, it has been noted that there exist no studies investigating the heart anatomically in deaths of cardiac and noncardiac origins. In this respect, it can be said that the findings of the present study are quite significant and the data from other researchers which will be used for discussion are rather limited. Other studies presented without basing the data regarding the papillary muscle on the cause of death may therefore lead to misleading comments.

In this study, the mean age of the cases was 40.9 years, a young age, unlikely to be affected by the changes that occur later in life. With aging, the tricuspid valve, like other tissues, changes; this change consists mainly of local fibrous thickening of both the papillary muscle and chordae. The right ventricular cavity becomes smaller as we grow older, and consequently, less area becomes available in the left ventricle to accommodate the tricuspid valve leaflets and chordae. The leaflets then contact one another abnormally during ventricular systole and focal fibrous thickening results. Sato and Shimada[22] demonstrated that Tenascin and collagenous matrix components showed a notably higher accumulation of elastic fiber concentration in the age group of 80s-90s than in that of 20s. This distribution and content of these extracellular matrix changes are related to changes in the mechanical performance of the papillary muscle due to age. The distribution of fibrous-elastic fibers differing between the outer and inner layers of the atrioventricular reflects their functional role in the opening and closing of the valve leaflets.[23] Extracellular matrix components may be expressed during aging and by altered stress. Sato also demonstrated that young people have ringlike structures, older people do not. The ringlike structures may be reducing mechanical tightening, twisting and slackening of the chordae tendineae. Nerantzis et al[24] described 3 types of papillary muscle arteries based on their amount of smooth muscle cells in people under 30. They demonstrated the atherosclerotic process of the papillary muscle arteries which changed the structure of the papillary muscles. They also observed that, the fibrous tissue increased in the arterial wall with aging. Automobile accidents, airbags, external cardiac massage and perinatal anoxia are the leading causes of traumatic tricuspid valve regurgitation.[25-27] The right ventricle can be indirectly injured by sudden increase of intracardiac pressure from compression of the upper abdomen and crushing of right intraventricular pressure in an injury of valvular apparatus.[28] The most frequently reported injury is chordal rupture, followed by rupture of the APM and leaflet tear, primarily of the anterior leaflet.[21,29] In the acute phase of the injury, traumatic lesions may go undetected. In the chronic phase, many patients remain asymptomatic and others exhibit symptoms and signs of moderate to severe right heart failure.[14] In his clinical case, Bardy et al[14] reported that the patient suffered blunt chest and abdominal trauma suggestive of a direct and indirect injury of the right ventricle.[14] dos Santos et al[30]

reported ruptured chordae tendineae of the posterior leaflet of the tricuspid valve as a cause of tricuspid regurgitation following blunt chest trauma.[18,30] Scorretti[31] reported a case of traumatic rupture of the APM. The patient survived for 3.5 h after injury without showing any serious cardiac symptoms, and in the absence of external chest injury. Becker et al[32] defined tricuspid valve dysplasia as faulty development of chordae and papillary muscle and thickening or focal agenesis of valvular tissue in congenitally malformed tricuspid valves.[9] Mc Elhinney et al[2] reported a distinct entity in which tricuspid valvular regurgitation resulted from failure of coaptation due to short chordae tendineae tethering the septal leaflet. Asymmetric, thickened and shortened chordae tendineae of the septal leaflet is a distinct cause of tricuspid regurgitation that can be recognized with autopsy. In their study Oosthoek et al[33] reported on mitral valves with focalized attachment of cords. They demonstrated that in autopsy specimens asymmetric mitral valves with 2 papillary muscles were present, and one of the muscles was elongated, located higher in the left ventricle with its tip reaching the annulus. The valve leaflets could be directly attached to this abnormal muscle that received few chords or no cords in 3 hearts, or to one of the 2 papillary muscles; a malformation which "parachute like asymmetric mitral valve. "They also found only one true parachute mitral valve, that is, one having a single papillary muscle that received all cords.[33] Papillary muscle necrosis was found in neonates without congenital heart disease, who died and were autopsied.[16] Types of sudden deaths and traumatic lesions may be associated with minor abnormalities of papillary muscle. We think that little relationship with the SPM, and differences in shape and number can be associated with the cause of death. We believe that this observation will provide the anatomist with valuable information concerning the structure of papillary muscle in right ventricle. Our studies on this topic are still going on. Our next aim will be to increase the number of cases included and further investigate whether blunt chest trauma has an effect on changing the structure of papillary muscles. A damage to valves or papillary muscles may occur after a trauma, and ischemia may negatively affect valve functions and create losses in the functional capacity of the body, even further leading to death. Congenital variations are known to be potential candidates for mechanical trauma and ischemic condition leading to papillary muscle lesions. For this reason, a detailed examination of heart valves as well as papillary muscles should be carefully performed to clarify the reason for sudden death with no apparent course. In future studies, cardiologists, anatomists and physiologists should examine whether the greater number of papillary muscle in sudden deaths and traumatic cases compared to the other groups is a reason for the structure, and probably the function, of the papillary muscle. All of these should be kept in mind by the forensic medicine examiner studying death cases and investigating the origins of death.

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