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Electrocardiographic changes in cardiomyopathic Syrian hamsters (strain BIO 8262)*)

Elektrokardiographische Veränderungen bei syrischen Goldhamstern mit Kardiomyopathie

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With 2 figures and 6 tables

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Summary

Under ether anesthesia electrocardiograms were derived from Syrian hamsters (strain BIO 8262) suffering from cardiomyopathy and muscular dystrophy. In addition, ventricular weights and body weight were determined. Young hamsters – not yet showing morphological signs of the cardiomyopathy with the exception of possible left ventricular hypertrophy – demonstrated only a longer ventricular activation time than normal hamsters. With the onset of cardiac necrotization left axis deviation in frontal plane projection and right bundle branch blocks are developing in the cardiomyopathic hamsters followed by first degree atrioventricular conduction defects. During the late stage of the cardiomyopathy left bundle branch blocks are additionally arising, while left ventricular hypertrophy is disappearing. Since no overt heart failure is occurring in this strain of cardiomyopathic hamsters, gradual development of high degree conduction defects is assumed to terminate their lives.

The electrocardiographic pattern of the hamster cardiomyopathy fits partly into that of human primary as well as secondary cardiomyopathy. Nevertheless, it seems to form an entity of its own, as arrhythmias, higher degree atrioventricular conduction disturbances, typical signs of ventricular or septal hypertrophy, abnormal P and Q waves, ST segment and T wave changes are lacking.

Human cardiomyopathy is known to exhibit certain electrocardiographic features (2, 3, 4, 5, 7, 8, 11, 14, 19, 20, 22, 23, 25, 27, 28). It is of clinical interest whether the spontaneous cardiomyopathy in the Syrian hamsters is electrocardiographically comparable with human cardiomyopathy. Moreover, it seems of importance to describe distinct stages of the disease by a clinical routine method, as morphologic (21), biochemical (16, 17, 18) and hemodynamic (26) studies have already outlined the course of the hamster cardiomyopathy. Since nothing is known about the cause of death in the diseased animals, the question concerning electrical problems in their hearts should be clarified.

*) Mit Unterstützung der Deutschen Forschungsgemeinschaft

Materials and methods

a. Animals

The experiments were performed on hamsters of both sexes of the cardiomyopathic strain BIO 8262, which was developed through cross-breeding between females of the original myopathic BIO 14.6 line and healthy males of the BIO RB line (see 16). The animals are afflicted with a recessive autosomally transmitted disorder of the striated musculature and the myocardium. It is described as muscular dystrophy and cardiomyopathy (1, 13, 21). According to the classification in humans (6, 10, 15, 24) the cardiomyopathy of the hamsters can be labelled as a secondary one. Light microscopic and histochemical examinations of the hearts revealed multifocal coagulation necroses predominantly occuring in the left ventricular wall and septum (21). Healthy animals from a commercially available strain served as controls. All hamsters were kept under identical controlled housing conditions; normal laboratory diet (Ssniff-H-hamster chow) and tap water were offered ad libitum.

b. Experiments

In accordance with histopathological findings in the BIO 8262 hamsters (21), three age groups were established:

I. 29 to 32 days: no myocardial lesions
II. 64 to 86 days: progressive multifocal coagulation necroses, calcification mainly of myocardial giant cells
III. 231 to 280 days: reduction of the number of new lesions, scar formation with calcification.

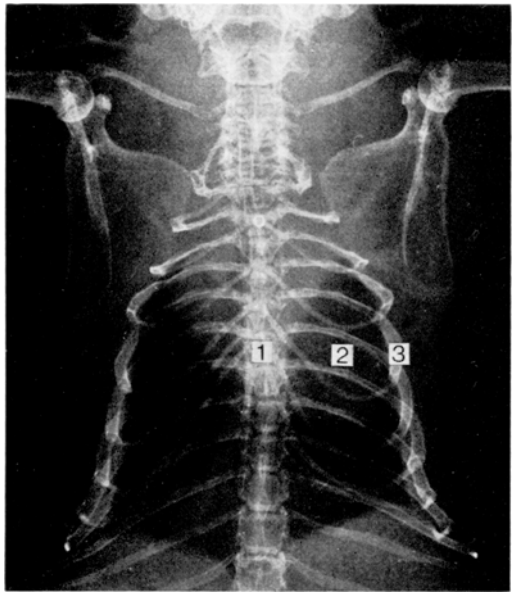


Fig. 1. Chest X-ray of a 256-day-old cardiomyopathic female hamster. Into point 1 (sternal line 5th intercostal space, about end of sternum), point 2 (midclavicular line 4th intercostal space), point 3 (midaxillary line 4th intercostal space) electrode needles were placed subcutaneously for electrocardiographic tracing. These points are demonstrated in their relation to the hamster heart.

Prior to the electrocardiographic tracing the hamsters were anesthetized with ether and fixed on a board in supine position, their extremities extended by rubber bands to the four corners. Needle electrodes were placed subcutaneously into the 4 extremities and on the thorax. The bipolar standard leads I, II, III, augmented extremity leads aVR, aVL, aVF and the unipolar precordial leads derived from the 4th resp. 5th intercostal space in the sternal, midclavicular and midaxillary line (fig. 1) were registrated. Anatomical studies showed the lead from the sternal line lying over the right and the leads from the midclavicular and midaxillary line lying over the left ventricular wall, the position of the ventricular septum being similar to that in human hearts. By repeatedly putting the electrodes on the animals identical electrocardiographic patterns could be seen.

For documentation the Siemens Mingograph 81 (6 channel jet ink writer) was used. The paper speed was 250 mm/sec. During the procedure the Ecg was monitored on an oscilloscope. It was only registrated, when the heart rate had stabilized just before the animals returned into the excitational stage.

After the electrocardiographic tracing the hamsters were sacrificed, weighed, and their hearts excised. The atria were carefully removed from the ventricles. Thereafter the right ventricular wall was exactly cut off from the septum. The total ventricular myocardium was then gently blotted on a gauze pad and weighed on a digital microbalance (Sartorius 2462). After removal of the right ventricular myocardium from the balance the remaining left ventricular tissue with septum was reweighed.

c. *Statistical evaluation*

From each of the parameters measured the mean value, additionally in healthy control hamsters with a 5 % probability of error the confidence limits of the mean values were calculated. When the mean value of a parameter of the cardiomyopathic animals lay beyond the confidence limits another population was postulated. The variables were presumed to be normally distributed.

Results

a. *General findings*

The most striking finding was sinus rhythm existing in all animals examined. Premature beats could not be observed. Atrioventricular conduction defects of second or third degree usually did not occur except when ether narcosis was too strong. This finding was also reported by *Hoenig* and *Mohr* (12), who were the first to derive electrocardiograms from hamsters in 1953. Atrioventricular blocks induced by ether quickly disappeared with decrease of depth of narcosis.

In figure 2a and b typical electrocardiograms derived from a normal and a cardiomyopathic hamster are demonstrated. Compared to normal human electrocardiograms in all hamster electrocardiograms a lower voltage could be observed. No difference existed between normal and cardiomyopathic hamsters in this respect. The presence of deep abnormal Q waves as observed in diffuse myocardial diseases and hypertrophic cardiomyopathies in humans could not be evidenced in the cardiomyopathic hamsters. Abnormalities of the ST segments and T waves were scarce, only very slight, and without any relation to the cardiomyopathy. The ST segments most frequently were isoelectric and the T waves of little height, the transition between both being fluent. Sometimes the T waves were

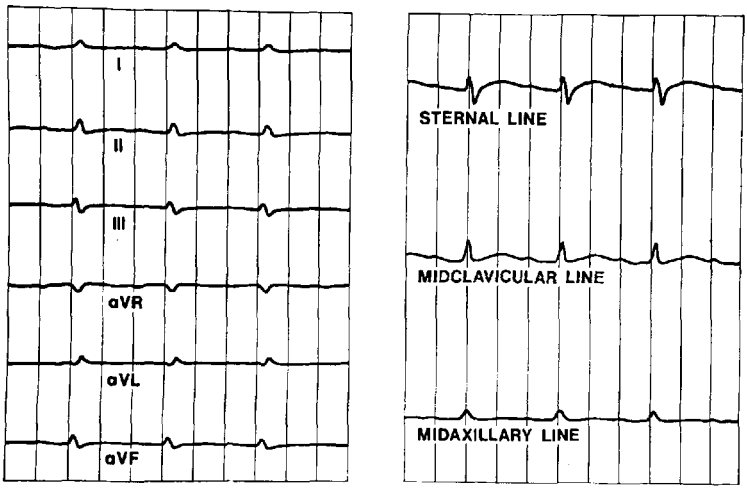


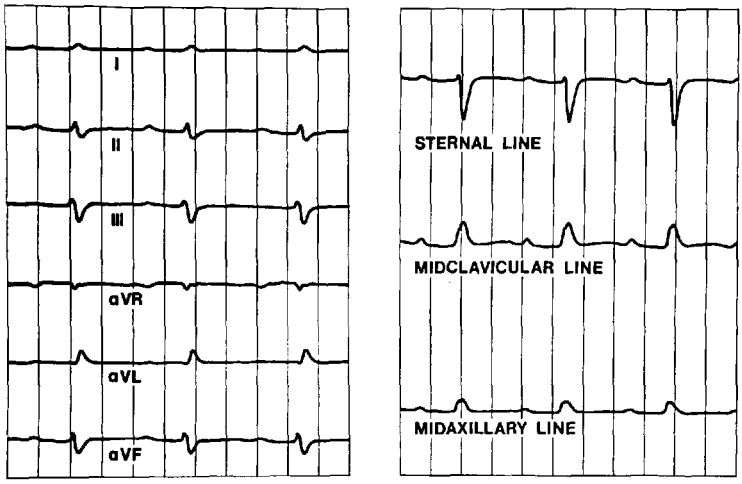
Fig. 2. a. Electrocardiogram of a 262-day-old male control hamster. The tracings are copied with black ink on translucent parchment paper for better contrast. Paper speed 250 mm/sec. The voltage is relatively low in comparison to human electrocardiograms. P and T waves are flat in the limb leads and in the mid-axillary line of the chest. They can be better discerned in sternal and mid-clavicular line. However, as it can be seen there the T waves are superimposed in their terminal parts by succeeding P waves.

straightened, an inversion could never be seen. Changes of the ST segment – if ever occurring – consisted of ascension. In addition, the end of the T waves could hardly be defined, as they mostly were superimposed in their terminal parts by succeeding P waves (fig. 2a and b). Hence, evaluation of the QT time was impossible.

b. Specific findings

In table 1 the behaviour of the direction of the electrical heart axis with age in frontal plane projection is demonstrated. In young hamsters of the first age group the electrical heart axis exhibits an intermediate position between $+30°$ and $+60°$ in most of the animals without difference between healthy and cardiomyopathic. With increasing age it shifts to the left side until $<-30°$ in many of the cardiomyopathic hamsters, whereas there is an opposite deflection to vertical position ($> +60° < +110°$) in most of the control animals with no further deviation in the oldest control animals examined.

The behaviour of the height of the R waves in the chest leads cannot be used to estimate the direction of the electrical heart vector in horizontal plane projection, as the amplitude of the QRS complexes varies with resistance depending on the depth of the electrode needles placed subcutaneously. For a coarse estimation of the dorsal deflection of the electrical heart axis the R/S relation in the sternal lead was taken (table 2). Whereas in the first two age groups there exists no difference in R/S relation between control and cardiomyopathic hamsters, in the third age



b. Electrocardiogram of a 257-day-old cardiomyopathic male hamster. The tracings are in the same manner copied from the original as those of the control hamster. Again low voltage as in the control animal. P and T waves are flat in the limb leads; P waves are a little more pronounced in the chest leads. T waves are again superimposed by succeeding P waves. Hence, QT time cannot be exactly determined. In contrast to the electrocardiogram of the control hamster there are deep S waves in leads II, III and aVF, the R/S relation in sternal line is much lower, the QRS interval is wider and a notch can be detected in the ascending limb of R in the tracing derived from the midclavicular and midaxillary line. These changes will turn out as being among the features of the hamster cardiomyopathy.

group the number of the cardiomyopathic animals with R/S relation less than 0.5 – even less than 0.2 – exceeds undoubtedly that of the control group.

In order to clarify the question whether the left and dorsal deflection of the electrical main vector of the older cardiomyopathic hamsters is caused by left heart hypertrophy heart weight, left and right ventricular weights, relation of left/right ventricular weight, heart index and body weight were determined (table 3). In the first age group similar heart weight and left ventricular weight does exist, however, lower right ventricular weight and increased relation of left/right ventricular weight is obvious in the cardiomyopathic hamsters. Together with lower body weight and increased heart index these findings point to relatively increased left ventricular weight in the cardiomyopathic hamsters in comparison to the controls. In the second age group heart weight, left ventricular weight and left/right ventricular weight relation and heart index are increased in the cardiomyopathic animals, right ventricular weight and body weight showing no difference between cardiomyopathic and healthy. As body weight of both hamster strains is similar increased left ventricular weight has now been proven directly. In the oldest age group only increased heart index is obvious in the cardiomyopathic hamsters, certainly as a calculative consequence of insignificantly de-

Table 1. Changing of the electrical hearts axis with age in normal and cardiomyopathic hamsters.

Age (days)	Strain	Range of electrical hearts axis in frontal plane projection				
		$< -30°$	$> -30° < +30°$	$> +30° < +60°$	$> +60° < +110°$	$> +110°$
29–32	BIO 8262	–	3	11	11	1
	Controls	–	4	13	8	1
64–86	BIO 8262	6	4	9	7	–
	Controls	2	1	4	16	3
231–280	BIO 8262	8	15	2	1	–
	Controls	3	1	5	13	4

The figures represent that number of animals out of a total of 26 in each group (BIO 8262; controls), which exhibit an electrical main vector (QRS axis) lying beyond resp. between the degrees given above.

Table 2. R/S relation in sternal chest lead in normal and cardiomyopathic hamsters of different ages.

Age (days)	Strain	R/S $\leq$ 0.5	R/S $\leq$ 0.2
29–32	BIO 8262	3	1
	Controls	0	0
64–86	BIO 8262	2	1
	Controls	2	0
231–280	BIO 8262	14	10
	Controls	4	1

The figures represent the number of animals of a total of 26 in each group (BIO 8262; Controls).

creased body and increased heart weight. Since left ventricular and body weight as well as the relation of left/right ventricular weight do not differ from the controls elevated left ventricular weight in old cardiomyopathic hamsters can be excluded.

The heart rate of all three age groups (table 4) exhibits no difference between cardiomyopathic and control hamsters, however, a tendency to decrease with age in both strains of animals cannot be denied. The range of the heart rates is in accordance with that found in golden hamsters by *Hoenig* and *Mohr* (12).

In table 5 the PQ, QRS interval and the ventricular activation time (VAT) in sternal and midaxillary line are demonstrated. In the first age group only the VAT in the midaxillary line is obviously delayed in hearts of the cardiomyopathic hamsters. In the second age group PQ interval is longer and VAT – both in sternal as well as midaxillary line – are also longer in the cardiomyopathic hamsters than in the controls, although the QRS interval is not widened. 9 resp. 4 out of 26 cardiomyopathic hamsters show a typical M-splitted (sternal line) right (RBBB), resp. notched left (midaxillary line) bundle branch block (LBBB) QRS complex (fig. 2b), whereas only 5 resp. 2 out of 26 control hamsters (table 6). In the third again as in the first age group VAT is only delayed in the midaxillary line of cardiomyopathic hamsters. But now, in contrast to the first age group the QRS interval is undoubtedly wider in cardiomyopathic hearts (table 5). 10 out of 26 cardiomyopathic hamsters exhibit the typical LBBB pattern whereas only 1 out of 26 control hamsters (table 6). The characteristic RBBB pattern can be seen in 12 resp. 8 out of 26 cardiomyopathic, resp. control hamsters. In all animals with RBBB or LBBB pattern VAT is equal to or longer than 10 msec. and the QRS interval lies between 20 and 28 msec.

Table 3. Heart weight+, ventricular weights, left/right ventricular relation, heart index and body weight in normal and cardiomyopathic hamsters of various ages.

Age (days)	Strain	Heart weight+ (mg)	Left ventr. weight (mg)	Right ventr. weight (mg)	Left/right ventricular weight	Heart index (mg/g b. wt.)	Body weight (g)
29–32	BIO 8262	148.85	118.05	30.81*	3.854*	4.03*	37.2*
	Controls	150.18 ± 11.63	115.49 ± 8.87	34.69 ± 3.2	3.382 ± 0.18	3.42 ± 0.11	44.4 ± 3.99
64–86	BIO 8262	272.26*	215.98*	56.28	3.881*	3.44*	79.6
	Controls	242.75 ± 17.21	190.27 ± 13.35	52.47 ± 4.67	3.686 ± 0.20	3.15 ± 0.14	77.9 ± 7.4
231–280	BIO 8262	402.85	322.12	80.74	4.428	3.75*	105.1
	Controls	392.22 ± 29.70	315.20 ± 24.33	77.02 ± 8.33	4.241 ± 0.35	3.49 ± 0.17	110.5 ± 8.24

The figures represent mean values of 26 animals in each group (BIO 8262; controls); additionally in the control groups the confidence limits of the mean values (5% probability).
+ = biventricular weight
* = difference statistically significant at the 5% level

Table 4. Heart rate of normal and cardiomyopathic hamsters of different ages.

Age (days)	Heart rate (min^{-1}) BIO 8262	Controls
29–32	445	440 ± 60
64–86	441	429 ± 48
231–280	422	411 ± 22

The figures represent mean values; additionally in the control groups the confidence limits of the mean values (5% probability). 26 hamsters in each group.

The PQ interval of cardiomyopathic animals of the third age group is again wider than that of the controls (table 5). While in the second age group only 3 cardiomyopathic hamsters display a PQ interval equal to or longer than 52 msec. (1° AV block), there are 14 in the third age group which do so. Only 1 control hamster of the second and 2 of the third age group do fulfill these criteria (table 6). Combination of RBBB with 1° AV block can be seen in 7 cardiomyopathic hamsters of the third age group, while combination of LBBB with 1° AV block does occur only in 2 cardiomyopathic animals (table 6). These possibly bilateral bundle branch blocks cannot be detected in the control hamsters. Combined RBBB and LBBB became obvious in 3 cardiomyopathic animals of the second as well as of the third age group, and the combination of 1° AV block (PQ interval 60 resp. 56 msec.) with RBBB and LBBB in 1 cardiomyopathic hamster of the same age groups (table 6). None of the control hamsters does reveal these high degree conduction defects.

Discussion

Until the sixth week of life the hearts of the cardiomyopathic hamsters appear light microscopically normal, while muscular dystrophy is already present since birth (21). In 29 to 32 day old cardiomyopathic hamsters relatively increased left ventricular weight of the prenecrotic hearts can be seen. However, electrocardiographically left ventricular hypertrophy cannot be detected unequivocally. Only VAT in midaxillary line is longer indicating a slight left ventricular conduction delay. Hence, left heart hypertrophy of minor degree might exist, as the weight studies and the delay of conduction point to this possibility.

During the necrotizing stage of the cardiomyopathy (64 to 86 day old hamsters) heart axis deviation to the left side in frontal plane projection becomes electrocardiographically apparent, whereas in normal hamsters the heart axis shifts to vertical position. Different deviation of electrical heart axis in horizontal plane projection cannot be evidenced between cardiomyopathic and control animals, when looking at the R/S relation. VAT in midaxillary as well as sternal line is delayed, however, the QRS interval again not being widened. As in this stage of the disease no significant difference of LBBB patterns does exist between cardiomyopathic and control hamsters (table 6), delayed VAT in midaxillary line might be in accordance with left ventricular hypertrophy. Weight analyses do now

Table 5. PQ and QRS interval and ventricular activation time (VAT) in sternal and midaxillary line of normal and cardiomyopathic hamsters of different ages.

Age (days)	Strain	PQ interval (msec)	QRS interval (msec)	VAT (msec)	
				sternal line	midaxillary line
29–32	BIO 8262	41.5	17.4	6.2	6.2*
	Controls	40.6 ± 1.31	17.9 ± 0.88	6.9 ± 0.89	5.3 ± 0.45
64–86	BIO 8262	45.8*	20.2	9.2*	8.3*
	Controls	43.5 ± 1.57	19.5 ± 0.95	7.5 ± 1.08	6.4 ± 0.86
231–280	BIO 8262	53.0*	24.1*	9.8	9.1*
	Controls	44.6 ± 2.19	21.0 ± 1.03	9.5 ± 1.49	7.2 ± 0.73

The figures represent mean values of 26 animals in each group (BIO 8262; controls); additionally in the control groups the confidence limits of the mean values (5% probability).
* = Difference statistically significant at the 5% level.

Table 6. Occurrence of electrical conduction defects in normal and cardiomyopathic hamsters at different age.

Age (days)	Strain	1° AV block	Bundle branch blocks					
					bilateral			
		PQ interval ≥ 52 msec	right VAT ≥ 10 msec QRS M-splitted in sternal line	left VAT ≥ 10 msec QRS notched in midaxillary line	right + 1° AV block	left + 1° AV block	right + left	right + left + 1° AV block
29–32	BIO 8262	–	3	–	–	–	–	–
	Controls	–	3	–	–	–	–	–
64–86	BIO 8262	3	9	4	–	–	3	1
	Controls	1	5	2	–	–	–	–
231–280	BIO 8262	14	12	10	7	2	3	1
	Controls	2	8	1	–	–	–	–

Each figure given above represents the number of animals out of a group of 26 hamsters.

clearly demonstrate increase of left ventricular weight in the cardiomyopathic hamsters. The delay of VAT in sternal line, however, cannot be attributed to right heart hypertrophy, as none of the measured weight parameters does agree with this possibility. Hence, 9 cardiomyopathic hamsters with RBBB pattern in comparison with 5 control hamsters showing this abnormality seem to demonstrate predominantly right ventricular conduction defect provoked by the cardiomyopathy. Additionally, slightly prolonged PQ intervals also display the onset of atrioventricular conduction defects during the course of the heart disease.

In old cardiomyopathic hamsters distinctly increased right or left ventricular weights could be excluded. However, an increased number of animals exhibited heart axis deviation to the left side in frontal and now additionally in horizontal plane projection. QRS interval and VAT in midaxillary line are prolonged. Since 10 out of 26 cardiomyopathic hamsters display a typical LBBB pattern in their electrocardiograms (see fig. 2b and table 6) occurrence of left ventricular conduction defects during this late stage of the cardiomyopathy must be assumed, while left ventricular hypertrophy is disappearing. However, extensive dilation of the left heart chamber does not seem to take place, since hemodynamic studies did not reveal elevated left ventricular enddiastolic pressure (26). Interestingly, VAT in sternal line now does not more differ from control hamsters. A possible explanation might be the increased incidence of unspecific RBBB in the control animals (table 6). Therefore, it becomes apparent that during the course of the cardiomyopathy RBBB are occurring prior to LBBB. Although, already during the second stage of the cardiomyopathy the PQ interval is prolonged (table 5), distinct first degree atrioventricular conduction defects mostly emerge in old cardiomyopathic animals (table 6).

From all these findings it can be concluded that during the prenecrotic stage of the cardiomyopathy there exists possibly slight left ventricular hypertrophy, which is regressive with increasing age, whereas right and left ventricular as well as atrioventricular conduction disturbances are successively arising. Hence, not only ventricular working myocardium, but also the conduction system gets affected by the inherited degenerative heart disease. However, the atrioventricular conduction system as well as the sinus node do not seem to get largely involved in the disease process, as heart rate is similar in cardiomyopathic and control hamsters (table 4) and atrioventricular conduction disorders of second or third degree as well as arrhythmias cannot be observed.

Since overt heart failure does not develop in the cardiomyopathic hamsters of strain BIO 8262 (26), electrical conduction defects of their hearts might keep them from longevity. The gradual occurrence of possibly bilateral blocks (table 6) might cease the life of the cardiomyopathic hamsters. Nevertheless, one has to be cautious with this reasoning, since nothing is known about the diagnostic as well as prognostic significance of the pathological electrocardiographic changes in hamsters. Histochemical fluorescence microscopical investigations on the conduction system of various mammals (golden hamsters, mice and rats) revealed differences in metabolism and innervation (9). The lifespan of the cardio-

myopathic hamsters is about 200 to 400 days; normal hamsters are reported to live for two to three years. This fact, however, could support the above made assumption.

Now, the question should be discussed, whether the spontaneous hamster cardiomyopathy resembles electrocardiographically human cardiomyopathy. As the hamster cardiomyopathy is combined with muscular dystrophy electrocardiographical findings occurring in humans suffering from muscular dystrophy and of the hamsters shall be compared. Arrhythmias, 2 : 1 atrioventricular block or changes of the heart rate could not be observed in the hamsters, as it is reported in progressive muscular dystrophy in man (4, 14, 22, 27, 28). Also tall right precordial R waves and deep Q waves in the limb leads and over the left precordium, taken as distinctive electrocardiographic pattern in Duchenne's progressive muscular dystrophy (22) were never seen. However, intraventricular conduction defects as reported by *Beckmann* and *Schmit* (4) in Duchenne's as well as limb girdle muscular dystrophy, could be also discovered in the dystrophic cardiomyopathic hamsters. The predominant electrocardiographic findings in the cardiomyopathic hamsters, as there are left and dorsal electrical heart axis deviations, PQ prolongation and intraventricular conduction defects are also features in patients with primary cardiomyopathy with and without hypertrophy (2, 7, 11, 19, 20, 25) and in patients with alcoholic cardiomyopathy (3). Arrhythmias, as tachycardia, atrial fibrillation and ventricular premature beats, changes of P waves, preexcitation syndrome, deep Q waves, signs of left heart hypertrophy, ST segment and T wave abnormalities, often seen in patients with primary and alcoholic cardiomyopathy (2, 7, 11, 19, 20, 23, 27 and 3), as well as the last three signs also noticed in Friedreich's disease (8), could never be evidenced in the cardiomyopathic hamsters. Nevertheless, it can be concluded that the electrocardiographic pattern of the hamster cardiomyopathy fits partly into that of human primary as well as secondary cardiomyopathy. However, it must be also stated that there do not exist highly specific and hence diagnostic electrocardiographic patterns in any form of human cardiomyopathy. The hamster cardiomyopathy itself seems to be more or less exceptional, since arrhythmias, higher degree atrioventricular conduction disturbances, typical signs of ventricular of septal hypertrophy, abnormal Q waves, P wave as well as ST segment and T wave changes are missing in their electrocardiograms.

Zusammenfassung

In Äthernarkose wurden bei syrischen Goldhamstern (Stamm BIO 8262), die an einer Kardiomyopathie und Skelettmuskeldystrophie leiden, Elektrokardiogramme abgeleitet. Zusätzlich wurden die links- und rechtsventrikulären Gewichte der Herzen und das Körpergewicht dieser Tiere bestimmt. Junge Tiere, die mit Ausnahme einer fraglichen Linksherzhypertrophie noch keine morphologischen Zeichen der Kardiomyopathie aufweisen, zeigen lediglich eine längere Dauer bis zum Beginn der größten Negativitätsbewegung. Mit dem Auftreten von Herznekrosen setzen eine Abweichung der elektrischen Herzachse in der Frontalebene nach links ein sowie das Auftreten von Rechtsschenkelblöcken, denen AV-Blöcke 1. Grades folgen. Im Endstadium der Kardiomyopathie treten noch zusätzlich Linksschenkelblöcke auf, während die Linkshypertrophie ver-

schwindet. Da sich bei diesen Tieren keine manifeste Herzinsuffizienz entwickelt, wird angenommen, daß die graduelle Entwicklung von bilateralen Blöcken ihr Leben terminiert.

Die elektrokardiographischen Zeichen dieser Hamsterkardiomyopathie passen teilweise sowohl zur primären wie auch zur sekundären Kardiomyopathie des Menschen. Dennoch scheint diese im Tierreiche vorkommende Kardiomyopathie in gewissem Grade eigenständig zu sein, da Arrhythmien, höhergradige AV-Blöcke, Zeichen einer Ventrikel- oder Septumhypertrophie, abnormale P-Wellen und Q-Zacken sowie Endstreckenveränderungen nicht beobachtet werden konnten.

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