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QUANTITATIVE STUDY OF THE INTRINSIC INNERVATION OF THE HEART IN ENDOMYOCARDIAL FIBROSIS AND AFRICAN IDIOPATHIC CARDIOPATHIES

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SUMMARY

A quantitative study of the intrinsic innervation of five hearts with Endomyocardial Fibrosis and nine cases of African Idiopathic Cardiopathies was made following Köberle's technique. In twelve hearts the neuronal count was lower than the average number of neurons in normal hearts. The average number of neurons in these cardiopathies was compared with the average found in Chronic Chagasic Cardiopathy. It was concluded that the pathogenesis of these African cardiopathies cannot be explained by the numeric diminution of the ganglion cells of the heart. Only further studies may establish the significance of the lower number of neurons found in these cardiopathies.

INTRODUCTION

The interest in the role of the autonomic innervation in the diseases is increasing since its importance was discovered in several morbid conditions. The best example of such a condition is Chagas' disease which in its chronic phase shows cardiopathy and "megas" — mainly of the esophagus and colon — due to parasympathetic dennervation [8].

A quantitative study of the intrinsic innervation of the heart in Endomyocardial Fibrosis (EMF) and African Idiopathic Cardiopathies (AIC) was done for four main reasons:

1) The etiology and pathogenesis of these cardiopathies are stil unknown. 2) There is no information about the intrinsic innervation of the heart in these diseases. 3) Parry's [21] statement that "EMF is a cardiac disease which is limited to the hot and wet regions of the tropics", the occurrence of EMF in expatriates (1, 4, 5, 7) and the existence of an acute initial illness and subsequent active disease, strongly points to an infectious agent. The fact that up to the time of working no microorganisms have been demonstrated does not exclude this possibility. In chronic chagasic cardiopathy, for example, parasites are only exceptionally found in routine examinations. Gerbaux & Rullière [6] advanced the hypothesis of an allergic mechanism caused by micro-filaria which could act even in the absence of the parasite. Thus, the possibility of a damage of the intrinsic innervation of the heart that could explain the pathogenesis of EMF is worthwhile to check. 4) The occurrence of idiopathic hypertrophy and dilatation of the heart, tachycardia and sudden, unexpected death in AIC which are also found in chronic Chagas' disease, suggest that both entities may have the same pathogenesis even if the etiology is different.

MATERIAL AND METHODS

Fourteen hearts were obtained from Africa — eight from Nigeria, five from Uganda and

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one from South Africa. Unfortunatly not all of them were in perfect conditions for this study, nine were already cut between the venae cavae and kept in formaline for more than one year. The age and sex groups and the diagnosis of the hearts are given on Table I. (In this table the original diagnosis are conserved, idiopathic cardiomegaly, heart muscle disease, South African endomyocardiopathy and endomyocardial fibrosis. The first three are named African idiopathic cardiopathies (AIC) in the present study but they probably represent the same morbid condition, described by BECKER et al. [2] and called South African endomyocardiopathy [3]. This is the reason that AIC are treated as one single group).

KÖBERLE'S [9] technique was used to assess the number of neurons. The area of the right atrium between both venae cavae was delimitated and cut and serial paraffin sections, seven micra thick were made. As a neuron is about 50 micra thick, only each seventh section was stained with haematoxylin and eosin and mounted. The neurons were counted only in these sections in order to avoid the same neuron to be counted twice. Thus, the figures on Table I, show the number of neurons in the area of the right atrium situated between the venae cavae.

RESULTS

The results are shown in Table I. The average number of neurons found in the cases of EMF and AIC are compared with the average found by KÖBERLE [10] in normal hearts and chronic chagasic cardiopathy in Table II.

DISCUSSION

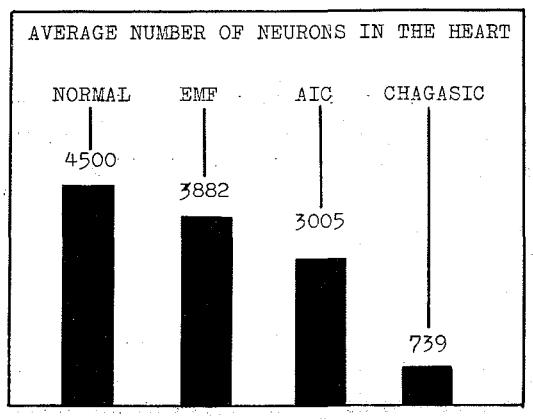
As it is shown in Table I, twelve African hearts had neuronal counts below the average normal. The experience in chronic chagasic cardiopathy has demonstrated that manifestations only occur in those hearts which have less than 75% (i. e. 3,375 neurons) of the normal average neuronal count (4,500 neurons) [11]. In this situation there are seven hearts: one EMF and six AIC. The other seven cases are not "significantly" below the average normal number. Table II, shows that although the average number of neurons decreases considering the order, normal — EMF — AIC — chagasic, there is a big gap between chagasic and African hearts. In KÖBERLE'S [10] series of 51 chronic chagasic hearts the highest neuronal count was 3,316 but the average only 739 (median

TABLE I

Case	Age	Sex	Diagnosis	no. neurons
U-1	48	m	IC	4857
N-1	51	m	HMD	4078
SA	32	m	SAEMC	3929
N-2	50	f	HMD	3054
N-3	42	f	HMD	3040
N-4	45	m	HMD	2687
N-5	42	m	HMD	2548
U-2	53	f	IC	1522
N-6	20	f	HMD	1329
U-3	21	m	EMF	4671
U-4	6	f	EMF	4173
U-5	34	m	EMF	3965
N-7	27	f	EMF	3740
N-8	25	f	EMF	2861

U = Uganda; N = Nigeria; SA = South Africa; IC = Idiopathic Cardiomegaly; HMD = Heart Muscle Disease; SAEMC = South African Endomyocardiopathy; EMF = Endomyocardial Fibrosis

TABLE II



value 480). Thus, even the *highest* figure in chagasic hearts (3,316) is lower than the *average* found in this study for EMF (3,882) and it is only slightly higher than 3,005 which was the *average* for AIC. It seems evident comparing the results of this work with the experience in Chagas' disease that if the diminution of neurons in EMF and AIC is real its significance is not the same as in Chagas' disease. It is questioned whether the lower figures found in the present work are meaningful because the number taken as average normal comes from hearts of non-Africans and there were only five cases in perfect conditions for the present study (U-1, U-2, U-3, U-4, SA). Considering that there is no difference in the number of neurons between normal negro and normal white Brazilians and our small experience do not indicate any difference in the neuronal count in normal African hearts; considering that the case U-2 was in good conditions and had the second lowest figure and considering that the 1,329 neurons of N-6 cannot be explained by technical difficulties, it is unlikely that the diminution of the number of neurons found in EMF and AIC is not real.

CONCLUSIONS

1) The pathogenesis of Endomyocardial Fibrosis and African Idiopathic Cardiopathies cannot be explained by the numeric diminution of the ganglion cells of the heart. 2) Only further studies may establish the significance of the lower number of neurons than the normal found in these African heart diseases.

RESUMO

Estudo quantitativo da inervação intrínseca do coração, na fibrose endomiocárdica e nas cardiopatias africanas idiopáticas

Realizou-se um estudo quantitativo da inervação intrínseca de 5 corações com Fibrose Endomiocárdica e 9 corações portadores de "Cardiopatia Idiopática Africana" seguindo a técnica padronizada por Köberle. Os resultados mostraram um menor número de neurônios do que a média encontrada em corações normais em doze dos catorze casos estudados. As médias da contagem neuronal nas Fibroses Endomiocárdicas e nas Cardiopatias Idiopáticas Africanas foram comparadas à média encontrada nas Cardiopatias chagásicas crônicas. Concluiu-se que a patogenia destas cardiopatias africanas não é explicável pela diminuição numérica dos neurônios intracardíacos. Sòmente estudos posteriores esclarecerão a significação da denervação parcial encontrada, na patogenia dessas cardiopatias africanas.

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