Intracardiac Electrocardiography in Fishes

In previous papers[1,2] the morphology of the epicardiac electrocardiogram in some Teleosts, Ganoids and Selachians has been reported. At the same time the pattern of the atrial and ventricular activation in the epicardiac surface of these animals has been established.

From such investigations it became clear that the excitation wave spreads radially through the auricular muscle, while the activation front in the ventricle follows an approximatively cranio-caudal direction without any appreciable difference among the various species examined.

In the present paper, the results obtained by recording the electrocardiogram from the atrial, ventricular and bulbar cavities of some freshwater Teleosts, of one Ganoid, and of two Selachians are reported.

Materials and Method. The investigations have been carried out on several specimens of *Cyprinus carpio* L., *Salmo irideus* Gibbons, *Ameiurus nebulosus* Lesueur, *Anguilla anguilla* L., *Acipenser sturio* L., *Scyliorhinus canicula* L., *and Scyliorhinus stellare* L.

A unipolar lead has been employed for the recording of the intracardiac electrocardiogram; the exploring electrode (a thin silver wire) has been introduced into the cardiac cavities through the bulbar wall, the indifferent electrode consisting of the Wilson's Central Terminal.

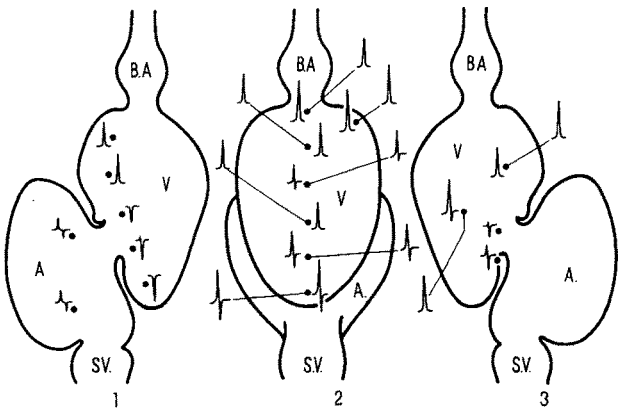

Fig. 1. The intracardiac electrocardiograms and the corresponding epicardiac tracings of *Anguilla anguilla*. B.A. bulbus arteriosus, S.V. sinus venosus, v ventricle, 1 right lateral surface, 2 ventral surface, 3 left lateral surface.

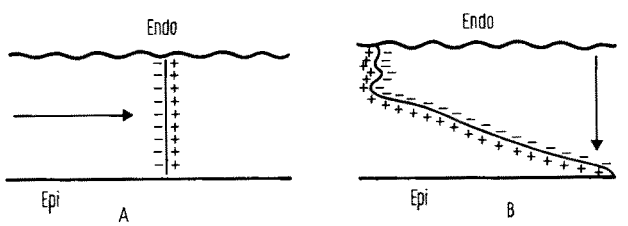

Fig. 2. The different types of activation in the dog ventricular wall (according to DURRER et al.[4]) and in the myocardial mass of the fish. Endo endocardial surface, Epi epicardial surface, A fish, B dog.

At the same time, a peripheral bipolar lead, homologous to the Standard limb lead D_{II} by EINTHOVEN, has been registered.

The intracardiac electrocardiogram and the epicardiac tracing topographically corresponding to it have been recorded at the same time.

Several points in the ventricle have been investigated; a smaller number of recordings were made in the auricular and bulbar cavities.

All the tracings have been registered by a Sanborn Twin-Viso electrocardiograph; different amplifications and paper speeds have been employed.

Results. Auricular cavity: in all the species examined an atriogram has been obtained, consisting of a positive, diphasic or negative deflection depending upon the position on which the electrode has been placed. Where simultaneous intra-auricular and epicardiac tracings could be registered, a close analogy between endocardiac and epicardiac patterns has been observed. Ventricular cavity: even the intra-ventricular electrocardiograms did not show any difference among the various species. As previously observed on the atrium, a close similarity between endocardiac and epicardiac patterns has been ascertained.

Prevailing negative deflections have been found near the atrioventricular funnel (it must be emphasized that an identical pattern of QRS complex has been observed on the epicardiac surface corresponding to the atrioventricular sulcus, above all posteriorly), whilst positive or diphasic deflections have been obtained elsewhere.

In almost all animals studied both endo- and epicardiac electrocardiograms started at the same time; only in a few specimens of *Scyliorhinus* the intraventricular tracings showed a slight delay (about 0.02) in regard to the epicardiac one. Bulbar cavity: the few endobulbar electrocardiograms did not show any remarkable difference from the externally obtained tracing.

Discussion. It is well known that in mammals (dog, cattle, horse, sheep, man) the intracardiac electrocardiogram consists almost constantly of negative deflections; furthermore the endocardiac tracing is registered earlier than the epicardiac one.

As a matter of fact, it is believed that the spread of activation in the mammal heart occurs from the inside outward; the excitation wave would first activate the subendocardiac layers and then spread outward.

From our experiments it can be believed that the spread of activation in the fish occurs synchronously in the whole muscle thickness as demonstrated by the similarity of the endocardiac and epicardiac patterns and by their simultaneous recording.

It can be supposed that the simultaneous activation of both endocardiac and epicardiac surfaces in the fish is very likely related to the lack of Purkinje fibers[3] in this animal. On the other hand, the early activation of the subendocardiac layers in the mammal is due to the presence, in this zone, of a well developed Purkinje network, which is not present in the subepicardiac and epicardiac layers.

Therefore we can think that in the fish the excitation wave spreads at the same time in all the thickness of the

[1] F. CHIESA, R. MARCHETTI, and V. NOSEDA, Arch. Sci. Biol. *46*, 1 (1962).
[2] R. MARCHETTI, V. NOSEDA, and F. CHIESA, Riv. Biol., in press.
[3] R. BORTOLAMI and L. BAZZACCO, Atti Soc. Ital. Sci. Veter. *6*, 330 (1952).
[4] D. DURRER, L. H. VAN DER TWEEL, and J. R. BLICKMAN, Amer. Heart J. *48*, 13 (1954).

cardiac muscle and that the activation front runs perpendicularly to the endocardiac and epicardiac surfaces.

Riassunto. Gli autori hanno studiato l'e.c.g. intracardiaco di alcuni Teleostei, un Ganoide ed alcuni Selaci. Le caratteristiche del tracciato endocavitario e di quello epicardico sono risultate assai simili; ciò fà pensare che l'onda di eccitazione si irradia, nel cuore dei Pesci, nel medesimo tempo in tutto lo spessore della massa miocardica e che il fronte di attivazione si sposta nello stesso tempo rimanendo normale rispetto alle due superfici.

V. Noseda, F. Chiesa, and R. Marchetti

Istituto di Fisiologia degli animali domestici e Chimica Biologica e Istituto di Zoologia, Università statale, Milano (Italy), May 15, 1962.

Coagulation Defect Following Non-Toxic Doses of *Echis viper* Venom

In their previous papers, the authors have drawn attention to the fact that the *Echis viper* venom, when applied subcutaneously in small doses to experimental animals, prolongs the coagulation time of the plasma[1]. To analyse this phenomenon more thoroughly, the effects of thirteen other snake venoms (*Vipera russellii, Vipera berus, Vipera amodytes, Naja naja, Bitis gabonica, Vipera lebetina, Crot. viridis, Crot. atrox, Crot. terrificus, Bothrops alternat., Agkistr. piscivorus, Agkistr. contortrix, Agkistr. halys.*) have been tested in the same manner, i.e. 24 h after the administration of doses ranging from 10 to 200 γ/200 g of body weight of the rat. The prolongation of the coagulation time has been ascertained in the *Echis viper* venom only. The experiments have been carried out on groups of white rats, each batch containing 10 animals of 200 g body weight. The tests were carried out 24 h after the application of the venom. A significant prolongation of the coagulation time developed already after a dose of 2 γ/rat. If 10 γ or more were applied, no clotting of the plasma could be observed even after 5 min. The changes were more obvious when assessing the thrombin time (0.1 ml of plasma, 0.1 ml of thrombin) than when determining the prothrombin time according to Quick. If 20 γ were administered to one animal, the thrombin time was significantly prolonged already 4 h after the application. When the coagulation was examined in dogs 24 h after a dose of 50 γ/kg, it was found that changes of the cofactors of the plasmatic thromboplastin were not involved in the defect. Both the level of prothrombin and its consumption were normal. The assumption is made that the coagulation defect was caused by changes which were due to the conversion of fibrinogen or to the fibrinolysis. The disturbed dynamics of the third and fourth phase of the coagulant activity is evident from the thromboelastographic pattern taken 12 and 16 h following the administration of 50 γ of toxin per rat (Figure 1).

The inhibition of the fibrin formation could be caused by a change in the fibrinogen molecule due to the inhibition of its polymerization or to the activation of the fibrinolytic system. The method employed for the determination of the fibrinogen level after its conversion into fibrin cannot be used to assess the effect of higher doses of *Echis* toxin, because under these conditions no fibrin is formed at all.

Dog plasma has, therefore, been analysed with the aid of electrophoresis 24 h after the application of 50 γ/1 kg. In all six experimental animals, no qualitative changes have been observed in the zone, corresponding to fibrinogen, which would deviate from the standard methodic error or the physiological variability (Figure 2). There are two possible explanations for this fact. The toxin could influence the molecular structure of fibrinogen in such a way that, though it does not change its electrophoretic migratory properties, it inhibits the conversion of fibrinogen into fibrin. This hypothesis will have to be corroborated by a closer analysis of the fibrinogen molecule after the application of toxin to experimental animals.

The active components of the toxin could act as an antipolymerase. The quantitative relations would, however, have to be taken into consideration. The direct inhibitory bonding of the toxin to the molecules of fibrinogen or its intermediary polymeres would have to follow stoichiometric laws where the ratio of the molecules in the reaction

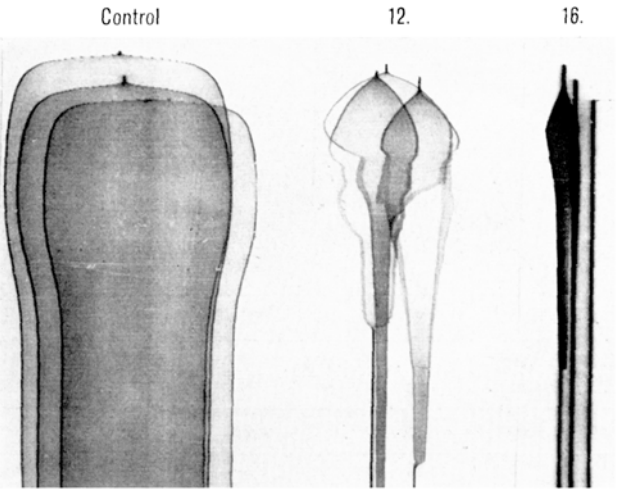

Fig. 1. Thromboelastographic pattern in control and experimental rats.

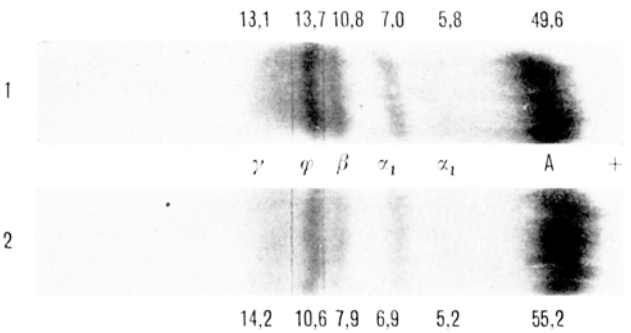

Fig. 2. Electrophoretic examination of dog plasma 24 h after application of *Echis* toxin. Number 1 control, number 2 24 h after application.

[1] F. Kornalík, Arch. exp. Path. Pharmak. *240*, 72 (1960).