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Toxicology

EFFECT OF ORGANOPHOSPHATE INSECTICIDE "OWADOFOS PŁYNNY-50" (FENITROTHION) ON BIOELECTRIC CHANGES IN CARDIAC MUSCLE OF EEL, *ANGUILLA ANGUILLA* L.

WPŁYW INSEKTYCYDU FOSFOROORGANICZNEGO "OWADOFOS PŁYNNY-50" (FENITROTHION) NA ZMIANY BIOELEKTRYCZNE MIĘŚNIA SERCOWEGO WĘGORZY *ANGUILLA ANGUILLA* L.

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The effect of an organophosphate insecticide "Owadofos płynny-50" (Fenitrothion) is shown in eel as a progressive bradycardia, elongation of ventricular changes (Q-T), and of an autonomic depolarisation of excitationgenerating centres (T-P). Intravenal application of catecholamines delays and moderates the ECG changes brought about by the insecticide.

INTRODUCTION

There is an urgent need for more studies on effects of insecticides as more and more humans and animals tend to be affected by insecticide-polluted waters (Janiszewski, 1973). Dissolved in stagnant and running waters, insecticides alter their physicochemical properties (Łuczak, 1966, 1971; Mac Donald a. all. 1969), hinder reproduction and growth of zoo- and phytoplankton constituting fish food (Cabejszek, a. Maleszewska 1970; Lis a.all. 1973; Ranke-Rybicka a. Stanisławska, 1972). In view of the world-wide opinion on the high toxicity of organochlorine compounds, organic esters of

phosphate acids showing a number of favourable properties such as a specific selectivity and fast degradability in natural environments, have been introduced. Owing to their excellent solubility in natural waters, organophosphate insecticides are easily absorbed by living organisms.

One of the most promising insecticides within the group of thiophosphate acid derivatives is Fenitrothion (0,0-dimethyl-0/3-methyl-4-nitrophenyl (-thiophosphate)), widely applied all over the world (Elson, 1972; Gocacka, 1971; IOSR Second List, 1961; Iatomi a.all., 1968; Symons, 1973; Wildish a.Lister, 1973). Compared to other parathion homologues, Fenitrothion is extremely selective to insects, its toxicity to mammals being lower than that of methyloparathion by the factor of 54. Outside a living organism, products of Fenitrothion degradation persist for a rather long time as 4-nitro-cresol (Bubień, 1971).

The data available indicate Fenitrothion to be moderately toxic (Nagahama a.all., 1960; Sumithion-1), or highly toxic (Bakuniak, 1970; "Zbiorowa Praca", 1970) to fishes. Bearing in mind a constantly growing use of Fenitrothion, a commercial preparate "Owadofos płynny-50" (Fenitrothion) was studied with respect to its effect on eel in terms of bioelectric changes in fish heart.

MATERIAL

The tests were carried out on 197 healthy eel individuals, length and weight of fishes examined being 35–40 cm and up to 300 g, respectively. Prior to the tests, the fishes were acclimated in aerated tap water in metal tanks for 24–36 hrs; the water temperature, pH, oxygen content, and total hardness ranged within 16–18°C 7–7.8, 9.2–10.2 ml/l, and 5.6–8.2 mval/l, respectively.

METHODS

The author's modification of Labat's (1966) electrocardiography was applied in the studies reported herein, indirect transverse precardial leads being used. Recording were made on an EK-2 Simplicard one-channel electrocardiograph. The ECG recording was in each case started as the breathing movements and heartbeat returned to normal, i.e., after the fishes with electrodes had remained 12–20 min. in water.

"Owadofos płynny-50:: (= Op-50) om a series of dilutions was applied underneath the water surface in the tank and the water was then circulated. The Op-50 concentration that reduced the heartbeat rate by one systole per minute and caused other constant ECG changes was regarded as a threshold value. Lethal doses (LD_{100}) were those concentrations killing the fishes after 2, 3, and 24 hrs of exposure ($LD_{100}2$, $LD_{100}3$, and $LD_{100}24$, respectively).

In order to study mechanisms of the OP-50-induced physiological compensation of the heart function, a pharmacologic analysis was performed by injecting the fishes intravenally with Regitine, Inderal, Adrenaline, Lovonor, and Isoprenaline.

The ECG recordings obtained with the constant shift of 25 mm/sec. were quantified, considering the length of ST, TP, PQ intervals and of Q-T, P-Q, and R-R phases in msec as well as the excitation frequency (no. of excitations per minute). These parameters were subject to a statistical treatment, mean values ($\bar{x}$) and standard error of the mean ($(S_{\bar{x}}$) being calculated.

RESULTS

Basically, the initial (physiological) ECG recordings of eels do not differ from recordings of other fishes and mammals (Fig. 2). The following elements are distinguished on an electrocardiogram: the auricular system (wave P, interval PQ, phase P-Q), the ventricular system (a combination of waves QRST, interval ST, phase Q-T), and a resting stage (interval TP). Measuring the R-R distances, a mean ventricular systole frequency was calculated as 49 ± 1.1 per minute, the range being 65.2–36.5 (Table 1). The waves revealed a high lability in relation to the isoelectric baseline; the wave R was always positive and present in every record. Fig. 1 illustrates diagrammatically a classical pattern on an electrocardiogram.

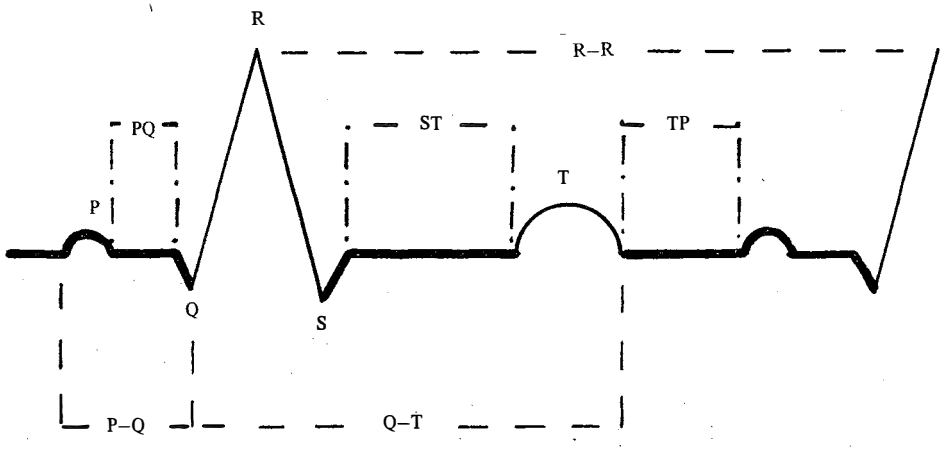

Fig. 1. A diagram of electrocardiogram: Waves: P,Q,R,S,T; Intervals: PQ,ST,TP; Phases: P-Q, Q-T, R-R

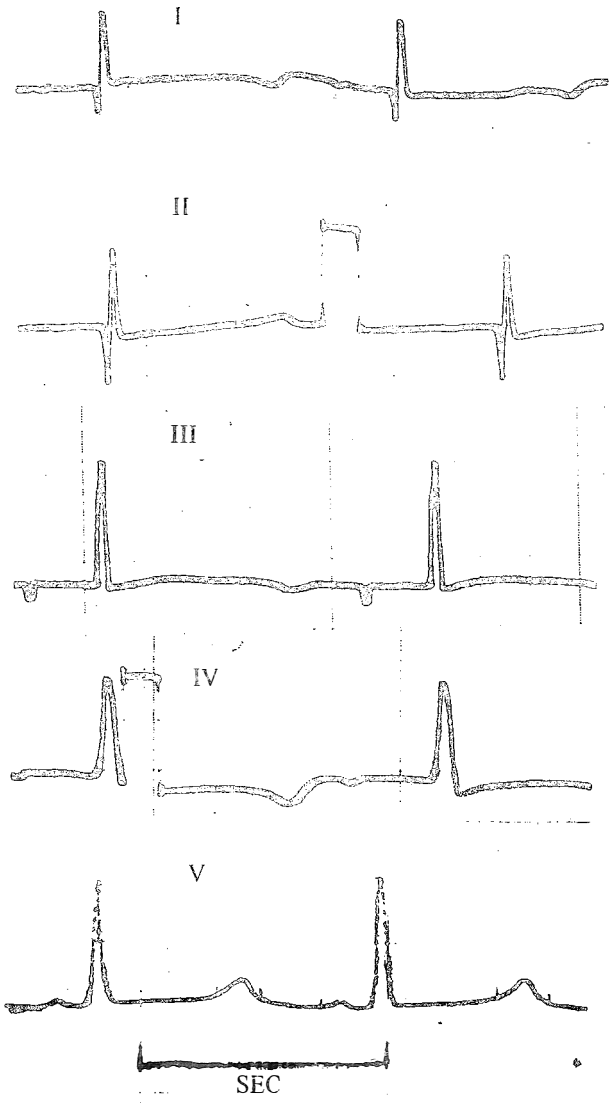

Fig. 2. Physiological electrocardiograms of aquarium-kept eels

Table 1

Mean values for ECG of aquarium-kept eels

No. of fish (n)	Excitation frequency (no/min.)	Intervals (msec.)			Phases (msec.)		
		ST	TP	PQ	Q–T	P–Q	R–R
40		40	40	40	40	40	40
Minimum (x)	36.5	628.0	508.0	240.0	1000.0	360.0	1643.8
Maximum (x)	65.2	280.0	140.0	40.0	488.0	120.0	920.0
Mean ($\bar{x}$)	49.2	438.6	255.1	132.6	714.3	218.9	1219.5
Standard error ($S_{\bar{x}}$)	± 1.1	±13.6	±18.1	± 9.1	±18.2	± 8.9	±28.4

I. ECG changes induced by Op-50

1. Exposure of fishes to a threshold concentration (0.001 ppm) results in a decreased systolic frequency, a higher amplitude of waves, longer intervals ST and TP as well as in longer phases Q-T and P-Q, and in a shorter interval PQ (Table 2). These changes resemble an inhibitory effect of the vagal nerve prompted by subjecting a fish to physical stimuli (a bradycardia with the simultaneous elongation of ventricular changes and depolarisation of ganglial cells). The increase in the wave amplitude reflects a higher electric potential of the cardiac cells (Fig. 3).
2. Lethal concentrations. The Op-50 concentrations of 6 ppm kill the fishes within 24 hrs ($LD_{100}24 = 6$). The first changes appear in the 18th second and proceed as a constant reduction of heartbeat rate and an elongation of the QRS duration. The 50 doses ppm impair the cardiac coduction which as of the 4th second shows up as a deceleration of systole, considerable arythmia, and differentials in the ventricular voltage. The fishes die our within 3 hrs ($LD_{100}3 = 50$).

Table 2

Changes in ECG of eels exposed to treshold concentration of Op-50 (0.001 ppm)

	Timing of ECG changes	No. of fishes	Excitation frequency (no./min.)	Intervals (msec.)			Phases (msec.)		
				ST	TP	PQ	Q–T	P–Q	R–R
Initial values	–	6	51.0	408.0	146.0	208.0	635.0	288.0	1176.5
Op-50 0.001 ppm.	1 min.	6	50.0	476.0	199.0	160.0	687.0	340.0	1200.0
	1 h	6	44.2	517.0	231.0	165.0	829.0	451.0	1357.5
	24 hrs	6	42.2	570.0	281.0	178.0	897.0	461.0	1421.8

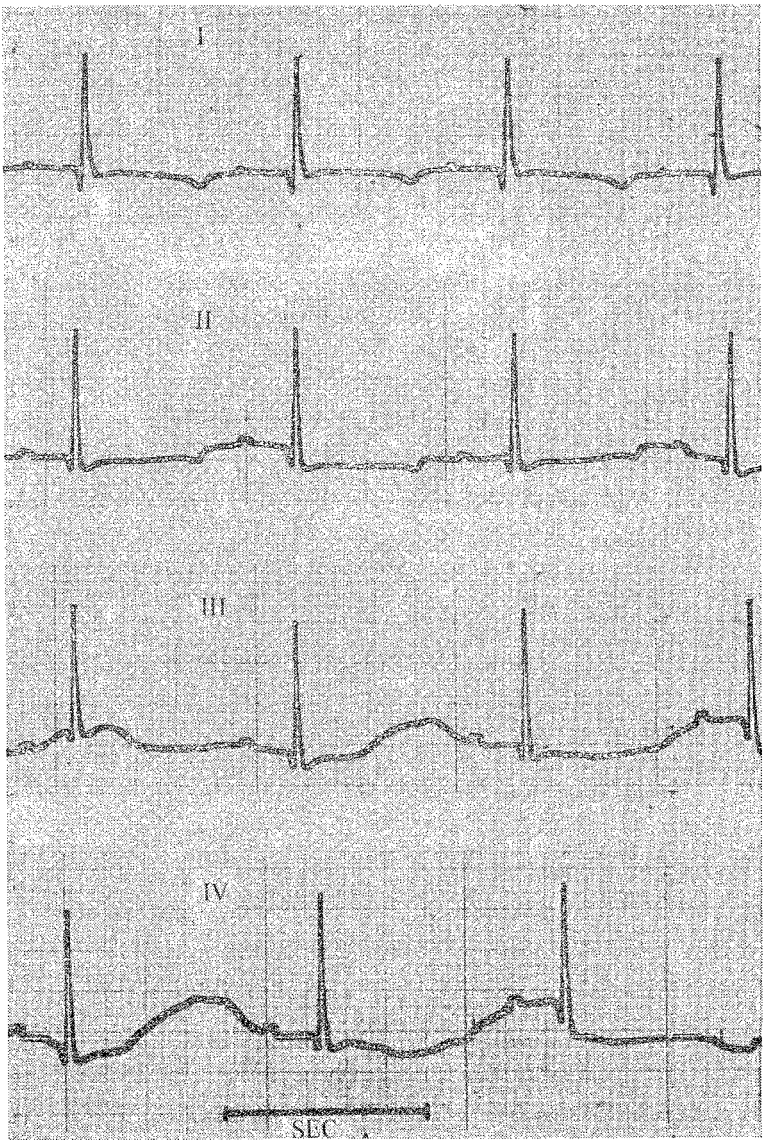

Fig. 3. Changes in ECG pattern of eel exposed to 0.001 ppm Op-50; I – initial recording, II after 1 min. III after 1h; IV after 24 hrs

Similarly, when using concentrations of 100 ppm, the first aberrant ECG is recorded in the 4th second, the complete absence of biocurrents being observed after 2 hrs ($LD_{100}2 = 100$) (Fig. 4). An instantaneous acceleration of ventricular systole frequency is recorded during the extinction of biocurrents.

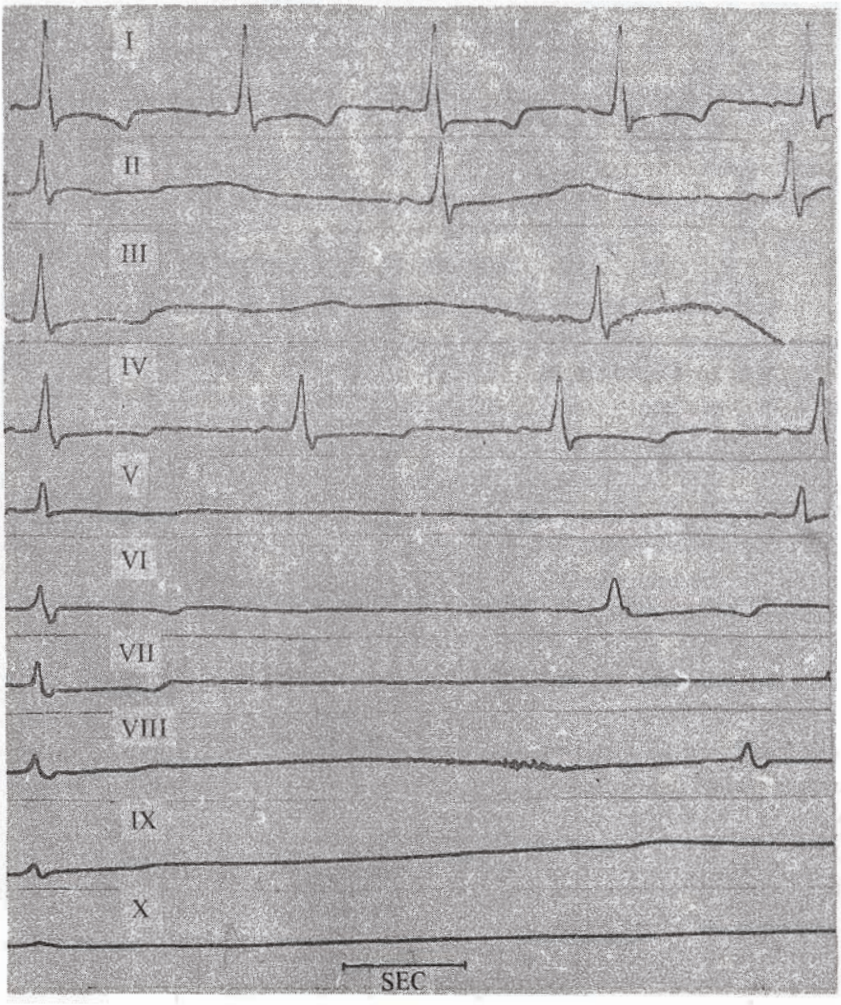

Fig. 4. Changes in ECG pattern of eel exposed to 100 ppm Op-50: I – initial recording
II – X recordings obtained during 120 min.

II. Shielding effects of adrenergic receptors antagonists and agonists

1. Regitine vs. Op-50. Pre-injected with Regitine (4 ng/kg) eels, when exposed to 0.1 ppm concentration of Op-50 showed a deceleration of systole frequency, a longer internodal conduction time and a lower amplitude of waves. The negative chrono- and dromotropic changes found to accompany the decrease in the electric potential, when compared to a response to the threshold dose and to the timing of the first ECG changes, indicate Regitine to inhibit the toxic effect of Op-50.

2. Inderal vs. Op-50. The first changes in the ECG of eels pre-injected with Inderal (40ng/kg) and exposed to 0.01 ppm Op-50 were seen after 4 min. of exposure as a deceleration of venous impulses and longer both TP and R-R, the waves being shallower. A higher concentration such as 0.1 ppm resulted in further changes occurring after 4 min. The exposure to 0.5 ppm Op-50 causes, after 2 min., an immediate slow-down of heartbeat, longer TP and R-R, and slightly shallower waves. Rapidly occurring negative chronotropic changes are much greater than those obtained without Inderal.
3. Adrenaline vs. Op-50. After 1 min. of exposure to 0.1 ppm Op-50 in water, eels-pre-injected with Adrenaline (0.4ng/kg) – showed a lower frequency of excitations, longer ST,TP, and R-R. A further weakening of excitations was recorded after 30 sec. of exposure to 1 ppm concentration of Op-50, a strong elongation of TP, Q-T, and R-R being seen too. In eels injected with 4ng/kg Adrenaline a 3 min. exposure to 1 ppm Op-50 results in a shorter TP and longer both PQ and P-Q, the excitation frequency remaining unchanged. It can be concluded then that the exposure to Op-50 of fishes injected with low doses of Adrenaline results in the negative chronotropy with a rapid bradycardia and a long-lasting depolarisation of excitation-generating centres. On the other hand a 6 min. exposure to 1 ppm Op-50 does not change the excitation frequency of fishes injected with 4 ng/kg concentration of Adrenaline, their ECG showing only slight alterations.
4. Levonor vs. Op-50. The first changes in the ECG of eels injected with Levonor (40 ng/kg) and exposed to 2 ppm Op-50 occurred after 3 minutes of exposure as a lower excitation frequency, elongation of ST, TP, Q-T and R-R. The amplitude of waves remained unaffected. Fishes without any protective injections, when exposed to an identical dose of Levonor, show the first alterations in their ECG as early as after 25 sec. An increase in the Levonor concentration to 10 ppm results in ECG changes after further 30 sec., which corresponds to the timing of appearance of the first changes at 1 ppm Op-50 without Levonor.
5. Isoprenaline vs. Op-50. Pre injected with Isoprenaline (4 ng/kg) eels, when exposed to 0.1 ppm Op-50, exhibited first symptoms of the insecticide's effect after 2 min. as alterations in the wave amplitude. Intervals and phases remained unchanged. Addition of 0.5 ppm concentration of the insecticide to the water manifested itself as an immediate change in the ECG waves, the excitation frequency being clearly reduced after 5 min.

When analysing the fish ECG alterations before and after the treatment with adrenergic synthetics, the latter were found to exert a favourable influence on the course of bioelectric events in heart of eels exposed to the insecticide (Fig. 5). Such adrenergic blockers as Regitine and Inderal show a weaker shielding effect then the agonists such as Adrenaline, Levonor, and Isoprenaline.

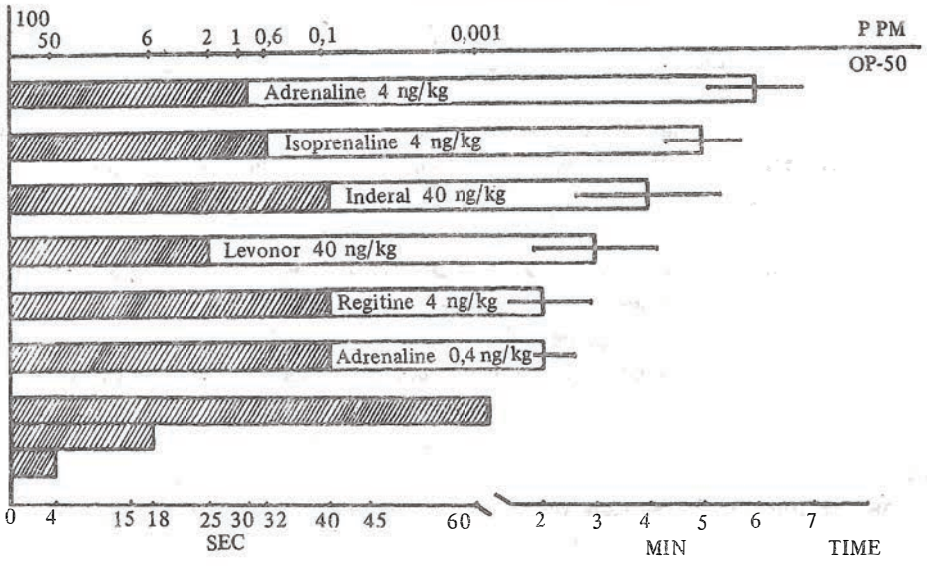

Fig. 5. Timing of occurence of negative chrono- and dromotropic phenomena on exposure to Op-50 prior to (dark fields) and after (bright fields) adrenergic treatment

DISCUSSION

The electrocardiographic technique applied in this study proved useful in assessing the degree of toxicity of the insecticide tested to eels. The fish heart gives a clearly negative chronotropic response to physical and chemical stimuli, which is explained as being caused by the depressing action of the vagal nerve (Belaud, a.all. 1969; Lutz, 1930; Randall et Stevens, 1967). The present studies have confirmed that the vagal bradycardia is accompanied by an increase in the electric potential, this being probably a compensating effect in the cardiovascular lining (Arniczyn, a.all., 1969; Cabrera a.Monroy, 1952; Randal a.Stevens, 1967) although there are suppositions that changes in the electric potential should not be too strictly identified with the cardiac muscle contractions (Lewartowski, 1969).

That acetylocholine, as a synaptic mediator, exerts a cholinergic influence upon the eels' ECG, is strongly advocated by similar changes brought about by physical stimuli, arecoline (a cholinergic ganglial venom), and low Op-50 doses, the latter compound being a cholinesterases inhibitor (Juszkiewicz a.Rakalska, 1968; Mott, 1957). However, one should not disregard the presence of considerable contents of adrenaline and levonor, reaching 50% of free catecholamines, in fish blood (Belleau, 1967; Bloom a.all., 1961; Jensen, 1961); this may evidence a humoral pathway of adrenergic stimuli conduction as the presence of adrenergic nerves in eel's heart has not been unequivocally proven. An

excess of acetylocholine in mammals results in the inhibition of excitation-generating centres, changes in the cardiac and smooth muscle tonus, intensified secretion, dilatation of blood vessels accompanied by a decrease in arterial blood pressure, disorders in heartbeat rate, and negatively dromotrophic phenomena (Ranc-Bukowska, 1970). Additioally, eels thus affected exhibit profound disorders in their electrolytic equilibrium, resulting in effusions in the abdominal cavity and infiltrational endema of viscera.

Zitko a. Carson, (1970) found a higher cholinesterases activity in brain of salmonids before spraying with Fenitrothion than after the treatment. A drop in the eel cholinesterases activity accompanied clinical changes caused by sublethal doses. Mac Donald a.Penny (1969) failed to detect any effect of commonly used concentrations of Fenitrothion on salmon juveniles and their food in water. Wildish a. Lister (1973) has proven that insects killed by Fenitrothion applied in doses used in practice cannot poison the fishes since the insecticide is lethal to insects in concentrations 3000 times lower than a sublethal dose for fishes. However, a decrease in amount of available food leads to a higher feeding activity of salmonids.

Combining the facts of: an intensified feeding (Sildish a. Lister, 1973), occurrence of compensative periods in the heart activity with the very favourable functioning of adrenal glands (Januszkiewicz a. Rakalska, 1968), a cholinergic effect upon the adrenergic control of macroenergetic phosphates of the heart (Garbuliński a.all., 1969) with the results obtained by Brzeziński (1973), it can be said that catecholamines tend to compensate for the stress caused by cholinesterases inhibitors. This finding is confirmed by the results of the present study: an intravenal injection of adrenergic compounds prior to the exposure to Op-50 delays and moderates the resultant ECG changes. Based on the literature available and the present stude, a conclusion can be drawn that eels possess an ability for a physiological compensation of environmental changes, this ability being expressed as a detoxicating action in Fenitrothion-polluted water.

CONCLUSIONS

1. "Owadofos płynny-50" (Op-50) can penetrate an eel's organism and alters the ECG in the manner identical to the effect of arecoline, which points out a similar mechanism of toxicity.
2. Lethal doses of Op-50 ($LD_{100}24$ = 6 ppm, $LD_{100}3$ = 50 ppm, $LD_{100}2$ = 100 ppm) result in a progressive bradycardia and lead to a complete cessation of cardiac biocurrents.
3. The egzogenic catecholamines applied (Regitine, Inderal, Adrenaline, Levonor, Isoprenaline) delay and moderate the ECG changes brought about by Op-50.

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WPŁYW INSEKTYCYDU FOSFOROORGANICZNEGO „OWADOFOS PŁYNNY-50" (FENITROTHION) NA ZMIANY BIOELEKTRYCZNE MIĘŚNIA SERCOWEGO WĘGORZY *ANGUILLA ANGUILLA* L.

Streszczenie

197 węgorzy o ciężarze ok. 30 dkg poddawano działaniu insektycydu „Owadofos płynny-50" (Fenitrotion) – 0,0-dwumetylo-0-/3 metylo-4-nitrofenylo/-tiofosforanu w warunkach akwaryjnych, rejestrując zmiany elektrokardiogramów w odprowadzeniu pośrednim, przedsercowym wentralnym.

Badany preparat powodował niezależnie od stężenia zmiany uznane za wagalne (postępująca bradykardia, wydłużenie zmian komorowych oraz depolaryzacji samoistnej ośrodków bodźcotwórczych), przy występowaniu okresowej tachykardii i wzrostu potencjałów elektrycznych komórek mięśnia sercowego. Czas zaniku bioprądów serca uzależniony jest od stężenia preparatu. Stwierdzono korzystny wpływ dożylnie wprowadzonych egzogennych amin katecholowych (regitina, inderal, adrenalina, lewonor, izoprenalina) na EKG węgorzy przebywających w środowisku skażonym owadofosem płynnym 50.

Й. Музыкевич

ВЛИЯНИЕ ИНСЕКТИЦИДА „ОВАДОФОС ЖИДКИЙ-50" (Fenitrotion) НА БИОЭЛЕКТРИЧЕСКИЕ ИЗМЕНЕНИЯ СЕРДЕЧНОЙ МЫЩЦЫ У УГРЕЙ Anguilla anguilla L.

Р е з ю м е

197 угрей ср. весом 300 г подвергнуто действию инсектицида „Овадофос жидкий-50" (Fenitrotion) – О,О-диметил-О-(3-метил-4-нитро-фенил)-тиофосфат в аквариумных условиях, фиксируя изменения электрокардиограмм в посредственном, предсердечном вентральном отведении. Исследований препарат вызывал, независимо от концентрации, изменения, которые считали вагальными (прогрессирующая брадикардия, удлиннение изменений желудочков сердца а также самопроизвольной деполяризацией центров автоматизма) при наблюдаемой периодичной тахикрадии и увеличению электрических потенциалов клеток сердечной мыщцы. Времия затухания биотоков сердца зависит от концентрации препарата. Установлено положительное влияние вводимых внутривенно катехоламинов (регитин, индерол, адреналин, левонор, изопреналин) на ЭКГ у угрей находящихся в среде загрязненой инсектицидом „Овадофос жидкий-50".

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