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Selenium Poisoning in the Chick Embryos

Zatrucia selenem u kurzych embrionów

Отравление селеном у эмбринов куриц

Selenium is an element found at different concentrations in soils, air and water. It is of particular interest because selenium is both toxic at high levels and essential at lower levels to organisms. The toxicity of selenium has been known since 1930, when studies of disorders observed among cattle and horses in USA, revealed that they were associated with ingestion of wheat or forages containing selenium (5). The pathology in acute toxicity is widespread necrosis and hemorrhage. The death is probably due to hypoxia secondary to these lesions in lungs. Manifestations of chronic toxicity are often species dependent and related to the form and amount of selenium ingested. In almost all species the liver is affected and cirrhosis develops. Cardiomyopathy is frequently found, loss of hair and sloughing of hoofs occur (1, 5, 9).

Teratogenic effect of selenium has been known in chickens. It has been recently reported that a higher number of abortions than expected were observed in group of laboratory animals which had been given selenite (3, 4).

As has been shown by Thompson and Scott selenium is known to be an essential element for the growth and development of chickens. Selenium deficiency was also shown to decrease both the hatchability of fertile eggs and viability of newly hatched Japanese quails. There is ample evidence suggesting a minimal dietary level of selenium for optimum growth and development in chickens. However, very little information is available detailing the level of selenium necessary for maximum fertility and hatchability in breeder hens (10).

It was found that injection of sodium selenite increased embryolethality and produced malformations in the survivors on the 2nd and 3rd day of incubation.

MATERIAL AND METHODS

Fertile chicken eggs were incubated at 38°C. Sodium selenite was dissolved in a buffer physiological saline and injected by aseptic technique into the yolk sacs at doses of 0.002 and 0.005 mg Na_2SeO_3 per egg. The injections were made on the 2nd, 3rd, 8th and 11th day of incubation. Control eggs were injected with equivalent volume of phosphate buffer. All eggs were injected only once. The living and dead embryos were removed from eggs and examined for the presence of malformations. Sections from the liver and heart were stained with hematoxylin and eosin for histological examinations. Sections from the heart were used for electron microscopical examinations.

RESULTS AND DISCUSSION

A distinct embriolethal effect of sodium selenite was observed in all groups tested. The toxicity of selenium was the highest when it was injected in eggs on the 2nd day of incubation. However, for embryos injected with selenium on the 8th and 11th day the mortality was significantly lower than for those injected on the 2nd and 3rd day of incubation. The mortality rate after injection of 0.005 mg Na_2SeO_3 on the 2nd day was 60%, on the 3rd day — 40%, on the 8th and 11th day — 10%.

Malformations in selenium treated embryos were manifested by microphthalmia (Fig. 1), the absence of an eye (Fig. 2), abnormal development of legs and feet (Figs. 1, 3, 4) and everted internal organs (Fig. 4).

The pathological changes observed in the internal organs were: congestion and parenchymatous degeneration in the liver and heart (Figs. 5, 6) and focus necrosis in the myocardium (Fig. 7). Enlarged cysterns of sarcoplasmatic reticulum were noticed in the myocardium after injection of 0.002 and 0.005 mg Na_2SeO_3 in comparison to control (Figs. 8, 9, 10).

Decreased body size and other teratogenic effects of sodium selenite may be due to the disruption of metabolic processes during development. Since selenium reacts with nucleic acids and protein synthesis, it is possible that the administration of selenium causes interference during the early stages of development. The biochemical mechanism of selenium toxicity has not been established yet. Recently *in vitro* studies have demonstrated glutathione dependent inhibition of amino acid incorporation into ribosomes by nanomole selenotrisulfide on critical enzyme sulfhydryl groups and formation of glutathione rendering the enzyme inactive (2, 11, 13). Rapid appearance of selenium in DNA and RNA synthesis and its association with tRNA will help explain the role of selenium in protein synthesis (6, 7).

Studies on rat liver showed that selenite did not block amino acid incorporation, provided that the supernatant used as the source of the enzymes is free of thiols. The addition of selenite to rabbit reticulocyte lysate resulted in inhibition of protein synthesis due to indirect inactivation of the initiation factor 2, while the elongation factor 2 was not effected. Also, in cultured cells an

inhibition of growth, protein synthesis and other functions were reported after administration of selenite. Since thiols are generally present in the culture medium, it is likely that the inhibition of protein synthesis in cells is partially due to the reaction products of selenite with these thiols. A reaction of selenite with intracellular thiols may also stimulate the formation of toxic products (8, 12).

The results of the present studies indicate that sodium selenite has embryolethal and teratogenic effect and causes pathological changes in the internal organs of chick embryos and chickens.

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STRESZCZENIE

Badano działanie selenu na embriony kurze. Selenin sodu wprowadzano do zalężonych jaj kurzych w ilościach 0,002 i 0,005 mg/jajo. Iniekcji dokonywano do pęcherzyków żółtkowych w 2, 3, 8 i 11 dniu inkubacji. Takie same ilości buforu fosforanowego wprowadzano do jaj kontrolnych. Obserwowano wpływ selenu na śmiertelność embrionów oraz wystąpienie zmian mikroskopowych w wątrobie i sercu. Zanotowano następujące zmiany: zmniejszenie ciężaru ciała,

niedorozwój kończyn, małoocze lub brak oka, wylewy krwi, zwyrodnienie miąższowe wątroby i serca oraz ogniska martwicy w mięśniu sercowym.

РЕЗЮМЕ

Исследовалось влияние селена на куриные эмбрионы. Селенин натрия был введен в завязанные куриные яйца в дозах 0,002 и 0,005 мг/яйцо. Инъекция была проведена в желточные мешки на 2, 3, 8 и 11 день инкубации. Такая же доза фосфорного буфера была введена в контрольные яйца. Исследовалось влияние селена на смертельность эмбрионов, а также появление микроскопических перемен в печени и сердце. Были отмечены следующие изменения: уменьшение веса тела, нарушение развития конечностей, микрофтальм или отсутствие глаза, пассивная гиперемия, паренгиматозное перерождение печени и сердца, а также фокусы некроза в сердечной мышце.

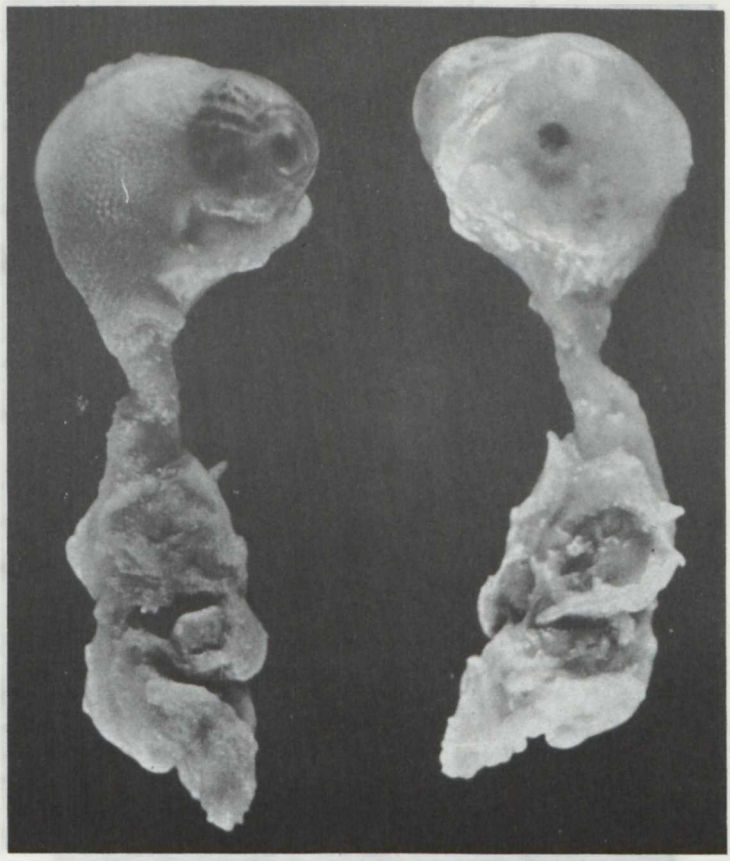

Fig. 1. A 16-day-old embryo. Microphthalmia and underdevelopment of legs

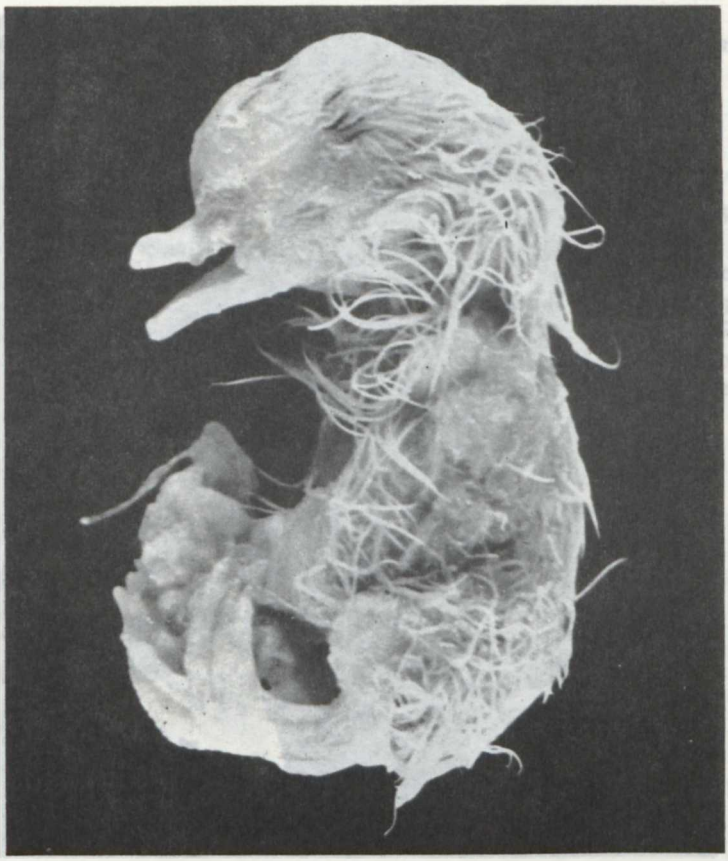

Fig. 2. A 1-day-old chicken. Absence of the left eye

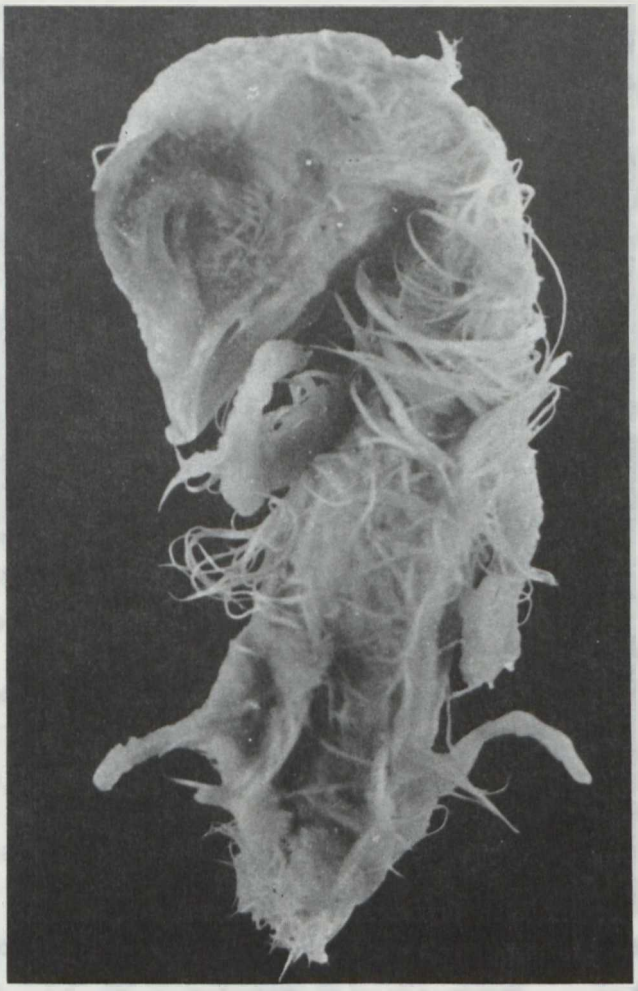

Fig. 3. A 19-day-old embryo. Underdevelopment of legs

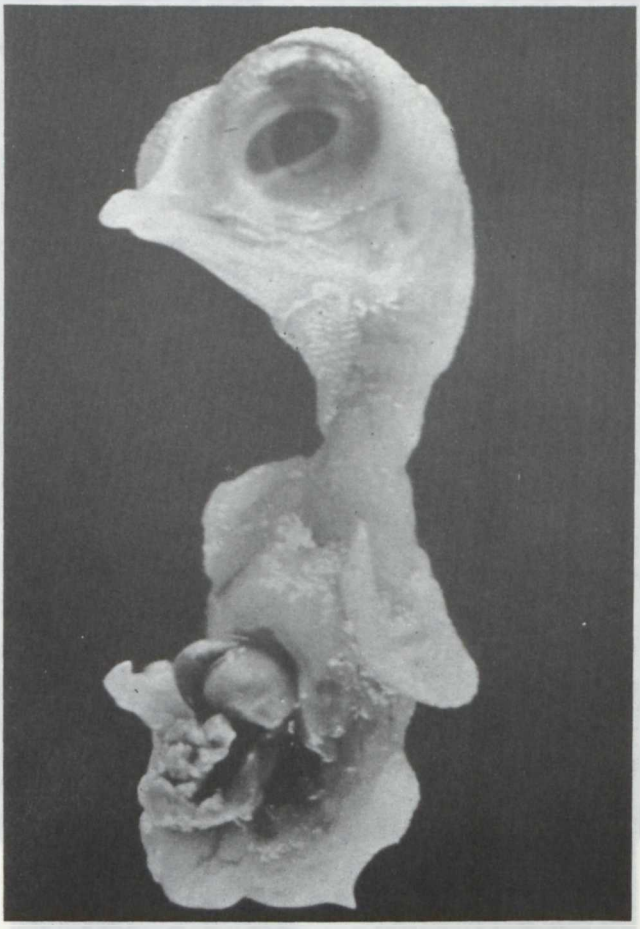

Fig. 4. A 20-day-old embryo. Underdevelopment of legs and of abdomen cover

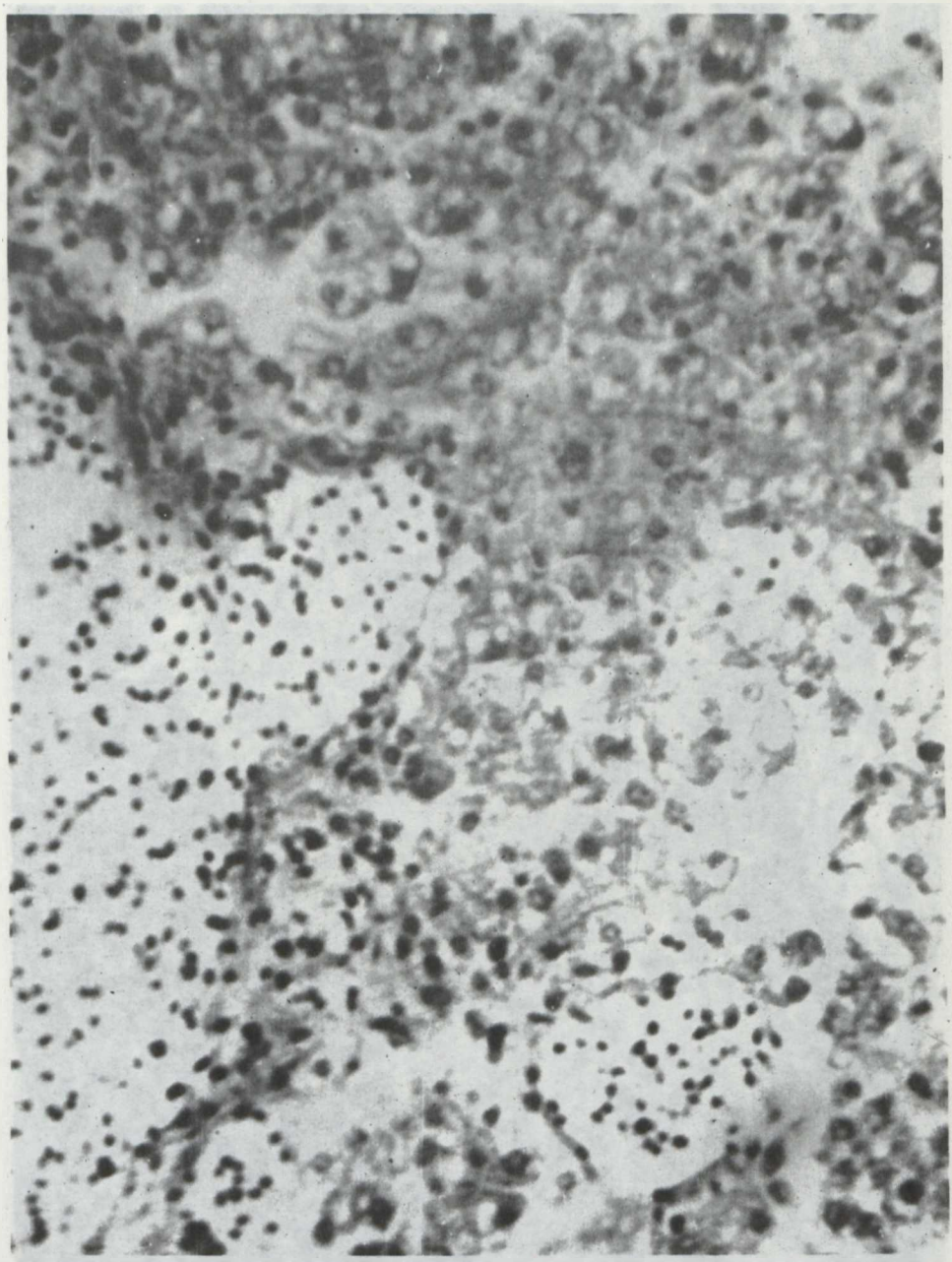

Fig. 5. Liver. Congestion and parenchymatous degenaration. Stain H + E. Magn. 240 ×

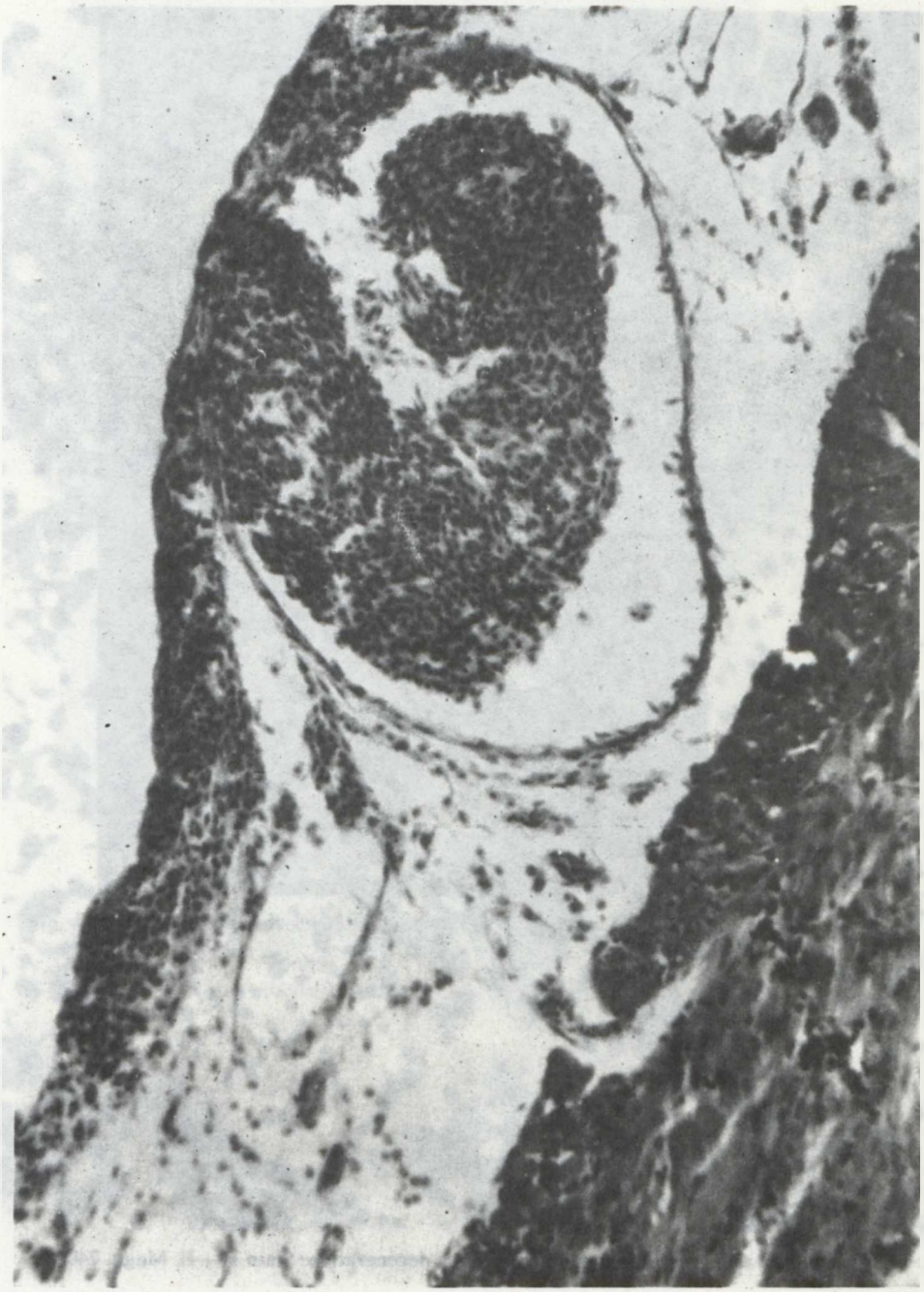

Fig. 6. Heart. Congestion and edema of pericardium. Stain H + E. Magn. 240 ×

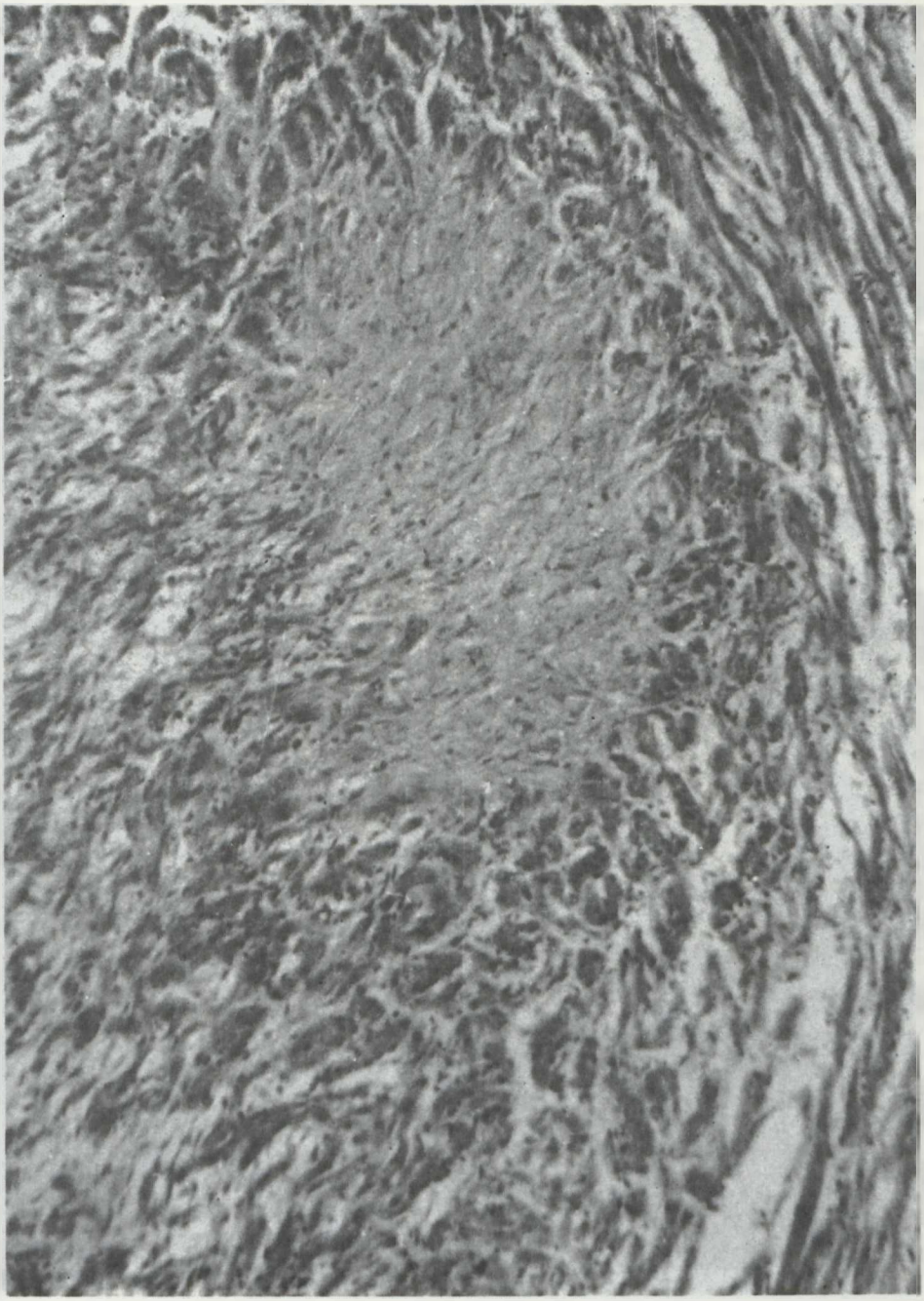

Fig. 7. Cardiac muscle. A necrotic focus. Stain H + E. Magn. 240 ×

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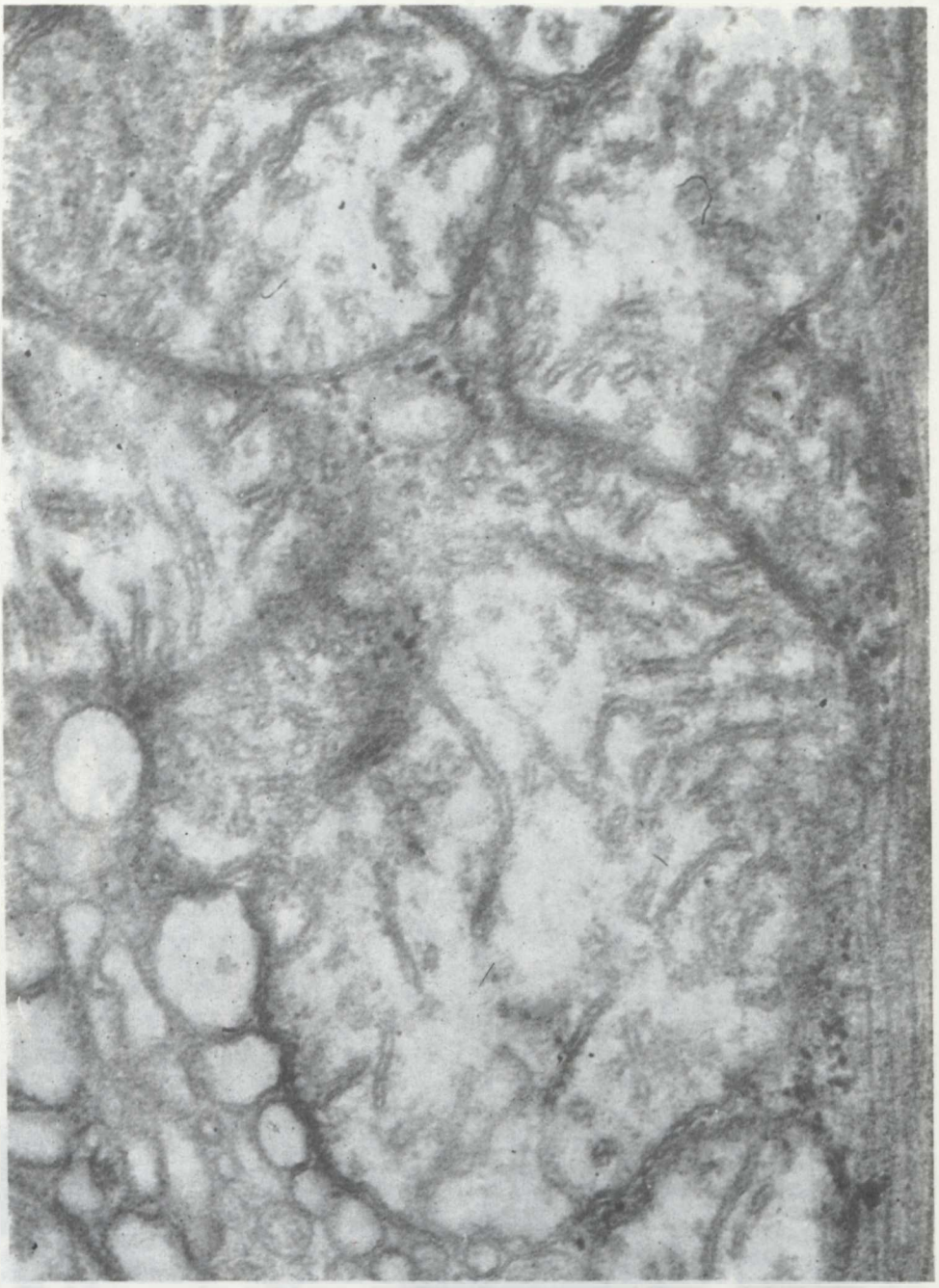

Fig. 8. Cardiac muscle cell. Control chicken. Sarcoplasmatic reticulum with small cysterns. Magn. 40 000 ×

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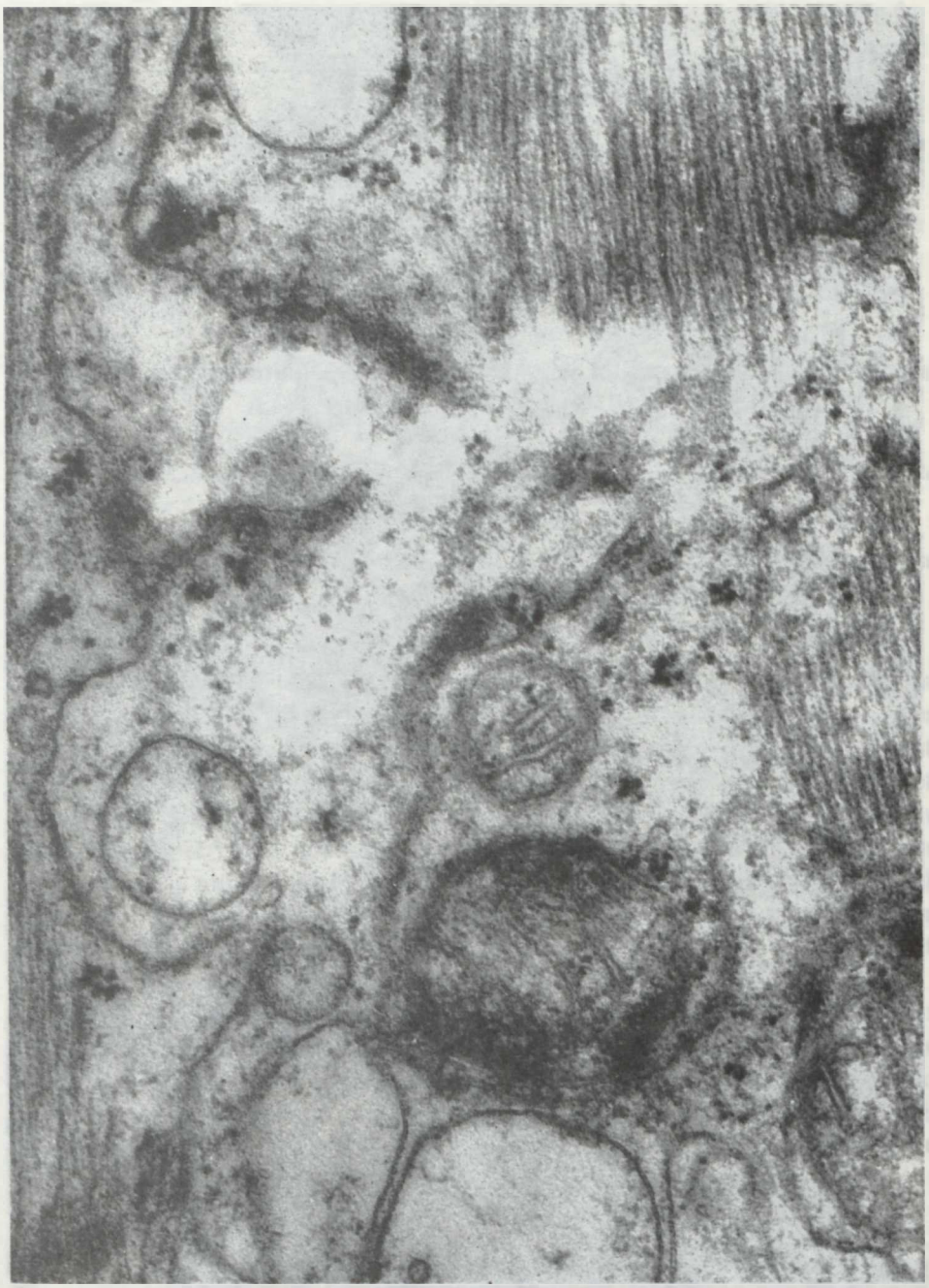

Fig. 9. A cardiac muscle cell after injection of 0.002 mg Na_2SeO_3. Enlarged cysterns of sarcoplasmatic reticulum. Magn. 40 000 ×

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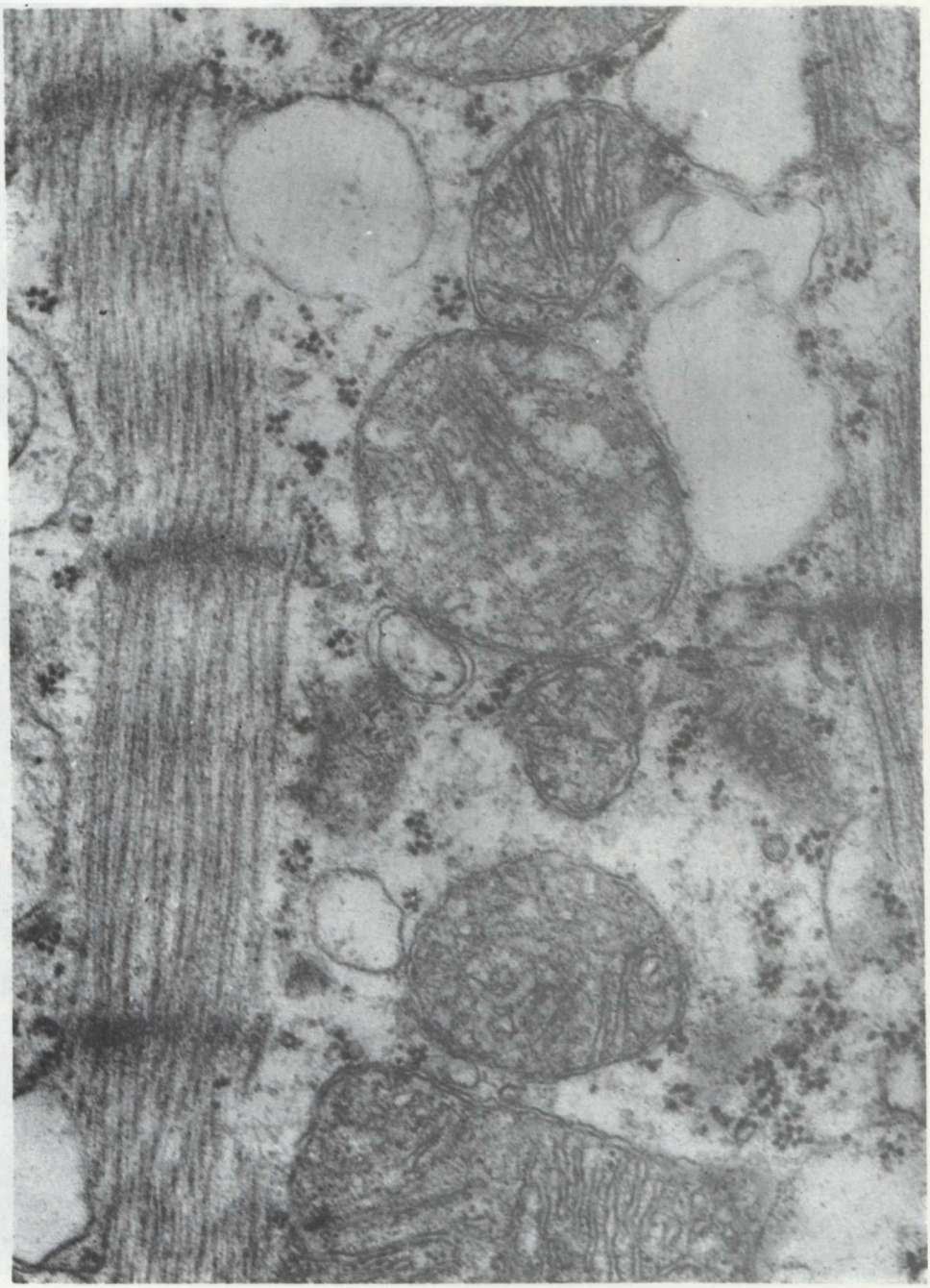

Fig. 10. A cardiac muscle cell after injection of 0.005 mg Na_2SeO_3. Enlarged cysterns of sarcoplasmatic reticulum. Magn. 40 000 ×

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