Table 3. Tunnel length and ratio of tunnel width to head capsule width in the 4 larval instars

	1st instar	2nd	3rd	4th
Tunnel length	4.0 mm	7.0	8.0	12
Tunnel width (TW)	0.26 mm	0.36	0.62	1.06
Head capsule width (HW)	0.19 mm	0.24	0.32	0.44
TW/HW	1.40	1.47	1.9	2.4

artificial kernels are as successful as the natural ones in feeding S. oryzae. No parthenogenesis is practiced by S. oryzae and virgin females do not lay any egg[20]. Nonetheless no failure in egg laying was encountered in any of the 30 pairs, which proves that the artificial kernels were as successful in producing efficient males as the natural ones.

Larval and pupal periods. 100 newly hatched larvae were marked with car paint and put in small holes made in food bands with 50 unmarked ones to study their duration. Results are indicated in table 2. Although temperature and RH were different in the present work, there is almost complete agreement between the figures tabulated and those given by Sharifi and Mills[4]. The tabulated figures also show that marking the larvae by paint may increase mortality, which, however, was high in the first instar whether marked by paint or not. The high percentage (70.2%) of mortality in larvae hatching on surface of food bands is attributed to inability of the hatching larvae to bite into the band surface; larvae hatching from eggs placed in holes in the food bands could bite into the walls of their holes. Their mortality percentages were far less (37.5%) than those of transferred first instar larvae (47–50%); this may indicate that normally mortality in the first instar is not as high as indicated in these experiments (47–50%). The occurrence of molting in the different instars was recognized by the absence of the marks from molted larvae and the presence of cast head capsules. Head capsules and tunnel widths of 10 larvae each of the 4 instars were measured (table 3). The ratio of tunnel width to width of the head capsule is not constant, as it increases slightly in the first 2 instars and abruptly in the third and the fourth. This may serve the future requirement of the next stages or it allows the fourth instar to turn head to tail in its tunnel.

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Progressive impairment in high energy phosphate pattern induced by intermittent coronary perfusion

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Summary. After continuous myocardial ischemia, ATP and CP are both depressed. A different pattern is exhibited in the course of intermittent coronary perfusion. The drop in ATP stores is not avoided, but progressive rise in CP contents is observed.

Ischemia has repeatedly been demonstrated to produce dramatic changes in myocardial function, cell structure and metabolism. Some data support the fact that short adequate coronary perfusion, associated with periods of ischemic arrest, could offer the advantage both of easy operative conditions and of reduced myocardial injury when compared to continuous aortic cross-clamping[2,3]. Such a concept may at first appear valuable. Coronary perfusion supplies substrates, namely glucose, and eliminates acidic components which may potentiate the deleterious effects of ischemia, particularly the loss of integrity of mitochondria[4]. Conversely reoxygenation has been shown to enhance some damage caused by ischemia, such as disruption of plasma membrane associated with enzyme release[5].

One of the effects of ischemia is the impaired ability to generate energy, a fact which is demonstrated by the decrease of high energy phosphate levels in the post-ischemic period. For Sakai[6], the more the myocardial ATP is decreased the more the rate of enzyme release is increased. In this study, the effect of intermittent coronary perfusion on the levels of ATP and CP during a series of ischemic and reperfusion periods was examined. A gradually increasing accumulation of CP was demonstrated.

Material and methods. 7 mongrel dogs, weighing 20–25 kg, were studied. Each dog was anaesthetized with pentobarbital (20 mg/kg). After tracheal intubation, ventilation was achieved with a volume controlled respirator. The animals underwent right thoracotomy through the fourth intercostal region. Heparin (3 mg/kg) was administered and the animals were submitted to extracorporeal heart-lung bypass. A bubble oxygenator with a heat exchanger primed with Ringer in order to reduce the hematocrit around 25% was used. The temperature was maintained at 37 °C and arterial pressure, pH, pO_2 and pCO_2 were monitored during the whole procedure. The heart was exposed in order to have an easy approach to the left ventricle avoiding any further handling of the heart. The aorta was cross-clamped for 15 min and then, by releasing the clamp, reperfusion was initiated for 3 min at a pressure of 80 mm Hg. During reperfusion, ineffective and irregular, heart beats could be

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observed. During ischemia and reperfusion, left ventricular myocardial biopsies were done with a drill technique described by Schwartz[7] on the left ventricular wall. The first samplings were made on the apex, following ones were performed moving up to base. The average weight of the samples was 50 mg. ATP and CP were assayed spectrophotometrically by the Fawaz method[8] and lactate by the fluorometric technique of Lowry[9].

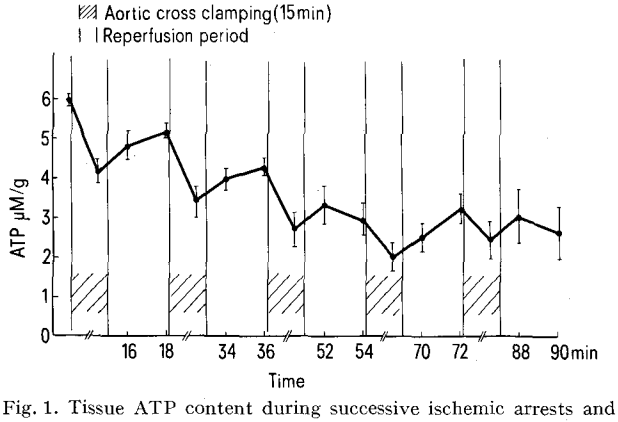

Fig. 1. Tissue ATP content during successive ischemic arrests and reperfusions. Each point represents the mean for 7 hearts and the bars represent SEM.

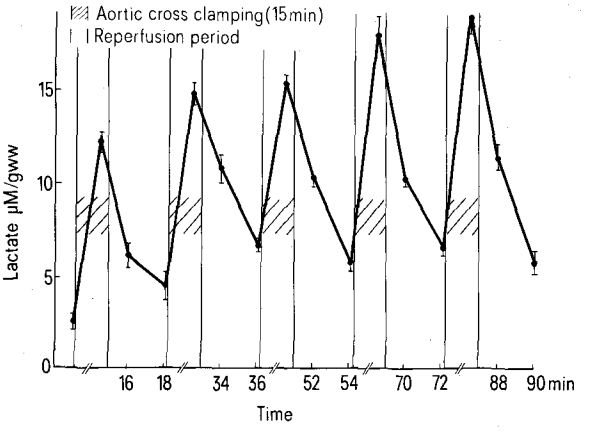

Fig. 2. Tissue lactate content during successive arrests and reperfusions. Each point represents the mean for 7 hearts and the bars represent SEM.

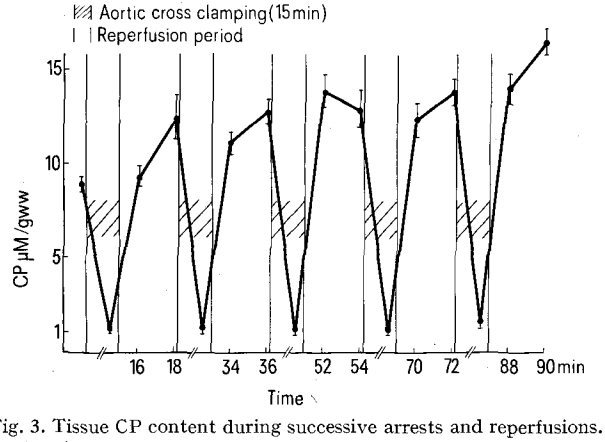

Fig. 3. Tissue CP content during successive arrests and reperfusions. Each point represents the mean for 7 hearts and the bars represent SEM.

Results. Figure 1 shows the drop in ATP during ischemic periods followed by partial resynthesis during reperfusion. This demonstrates that intermittent coronary reperfusion did not prevent ATP fall during ischemia. After 45 min, 50% of initial level has already disappeared. The average synthesis ranges at about 25% of the value before reperfusion. Figure 2 shows the evolution of tissue lactate. After 3 min, no return to initial state is achieved. This might indicate either incomplete washout and/or insufficient return to the aerobic state. This shows that acidosis will persist in the myocardial tissue despite reperfusion. Further experiments showed that 10 min at least were necessary to observe a return to basic conditions. It must be noted that glycolytic rate increased over the successive periods, since lactate production varied significantly from 9 µM/g to 13 µM/g wet weight at the end of the fifth ischemic period. The most striking fact is that during this procedure of myocardial 'protection', an unusual pattern of CP resynthesis was observed (figure 3). All values dropped abruptly during the ischemic period and the same low value of 1 µM was reached whatever the initial values. Resynthesis rapidly took place, leading to higher contents each time. So, at the end of 90 min, the initial value has doubled. In order to appreciate whether this phenomenon was transient, in some cases duration of reperfusion was lenghtened up to 30 min, but CP always remained at the level reached after 3 min.

In this work, it was shown that intermittent coronary perfusion is unable to preserve the cell ATP potential. These results are in strong agreement with the work of others. Such an impairment occurs between the 5th and 15th min[10]. The striking fact is the progressive increase in CP levels after each recovery period. At the end, it corresponds to the entire conversion of creatine into creatine phosphate[11]. Generally when CP was studied after reperfusion following longer ischemic arrests its level was found depressed[12,13]. Kübler has demonstrated a direct relationship between resynthesized CP and ATP at the end of arrest. Nevertheless, a rise in CP averaging 1 µM/g w/w over initial level after short periods of anoxia has been reported[14]. The reason why such a phenomenon occurs has not yet been demonstrated, but the fact that the heart is not working may be the cause. On KCl-arrested, well-arrested, well-oxygenated and empty hearts, Hassinen[15] and Lochner[16] have observed a moderate increase of CP, and Hearse[17] a sharp one. On

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all cases, the ATP-contents were normal. It has been suggested that such a CP storage is due to imbalance between production and utilization. Here the situation may appear different, since the hearts were neither fully empty nor completly arrested and the ATP levels were low. A blockage in the transfer between the 2 CP compartments is more likely[18]. If we refer to Lohmanns reaction:

$$ATP + C \rightleftharpoons CP + ADP.$$

The optimum pH in the direction of CP formation is 8.00 and 6.4 for the reverse reaction. After ischemia, the intracellular pH is lowered[19] mainly by accumulation of lactic acid. The washout curves presented here show that CP resynthesis is achieved at a time when high levels of this metabolite are still present. It then appears paradoxical that the results should favour the forward reaction. Concerning the mitochondria one may postulate either a local rise in creatine[20], or more probably the fact that ADP store is progressively reduced, implying its rapid utilisation as soon as it is generated for oxidative phosphorylation.

Nevertheless, this resynthesis of CP is indicative of an active oxidative phosphorylation as both this latter reaction and creatine kinase activity are linked. The inability to recover normal ATP contents may then be due to rapid degradation at the end-product stage of ATP rather than to mitochondrial damage as observed after longer periods of ischemia. So intermittent coronary perfusion seems to protect the mitochondria from the effect of anoxia. Whether this is beneficial to the heart is debatable. Kammermier[21] has observed a normal function on hearts with high CP and low ATP levels. In our experiments, hearts with such a high energy phosphate pattern failed.

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The function of the intestine in the pulmonate mollusc Helix pomatia L.

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Summary. Everted sacs of intestine from Helix pomatia do not actively transfer glucose, galatose, methionine, alanine, asparagine, aspartic acid or proline from the mucosal to serosal surfaces. The principal function of the intestine appears to be the reabsorption of water.

The molluscan intestine is usually a long and conspicuous organ[3] which a consensus of opinion, summarized by Yonge[4,5], considers to be a passive conduit concerned almost exclusively with the formation and elaboration of faecal pellets and their transport to the anus. This 'classic theory'[6] of the function of the molluscan alimentary canal, in which the intestine is assigned a prosaic role, has been reiterated – or not seriously questioned – in a number of recent reviews[7–10].

The principal evidence against this view has come from studies by Lawrence and his colleagues[11–18] on the chiton Cryptochiton stelleri where the intestine was found to actively absorb amino acids, sugars, organic bases and probably inorganic ions, by mechanisms coupled to cellular metabolism and showing similarities to those of the vertebrate gut.

In refuting conclusions from earlier work[19] on the chiton it was shown that, without supporting quantitative data, histological and histochemical studies are not adequate for a rigorous demonstration of nutrient absorption. Consequently, the findings of other studies, similarly based upon qualitative or semi-quantitative methods were also questioned. It has been proposed that absorptive mechanisms, similar to those in chitons, may occur in the gut of other molluscs[12], including pulmonates[20].

This paper considers the competence of the intestine of the pulmonate mollusc (Helix pomatia) to absorb amino acids and hexoses in vitro. There is a distinction to be made between absorption into the tissue, a general feature of living cells, and absorption into and across the gut wall, a function of tissue specialized for the assimilation of nutrients into the animal. Failure to recognize

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