piston and cylinder, and the method of interrupting the outflow of air, which causes the blood to be forced out of the balloon.

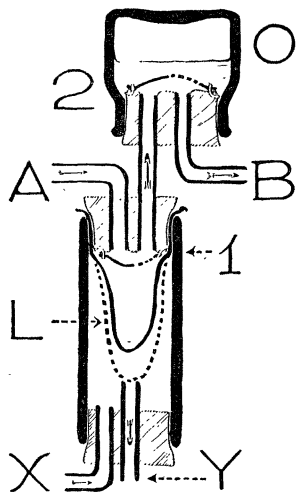

FIG. 1.

Compressed air flows from the main into the apparatus at X at a constant rate. Blood enters at A, flowing past the flap valve (1) and distending the balloon (L) forcing it down against the opening in the air exhaust tube (Y) and closing it off. A sudden increase in air pressure results and the tip of the balloon is held firmly against the opening in the exhaust tube. Then the blood inside the balloon is forced out past a second flap valve (2) into the bottle (0) which serves as an air cushion. As the hydrostatic pressure in the balloon (L) is decreased, the tip of the balloon is finally pulled away from the exhaust tube (Y) by the elasticity of the rubber wall of the balloon. Consequently, the air pressure in the apparatus is almost immediately released and air entering at the intake (X) again escapes from the exhaust (Y) at a constant rate. Within the range of its maximum capacity the speed of the pump is automatically adapted to the rate at which the blood is flowing in. When the inflow stops, the balloon does not fill and consequently the air exhaust (Y) remains open.

Flap valves, made of a portion of a toy balloon or, better yet, of dental rubber dam were found to be easily constructed and quite dependable. The rubber dam is sewed at its edges to the rubber stopper, being careful to allow enough slack so that blood flow is not impeded. As indicated in Fig. 1, the balloon (L) is slipped over the rubber stopper after the intake valve (1) has been completed.

Once the air exhaust tube (Y) is adjusted to the length of the balloon (L) and the optimum rate of air inflow determined, the pump works consistently with little attention. Our model pumps 600 cc per minute (about 10 cc per beat) against a hydrostatic pressure of 250 cm H_2O, with a pressure at the intake of about 20 cm H_2O.

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SPECIAL ARTICLES

TOXICITY OF NATURALLY OCCURRING ARSENIC IN FOODS

IT is well known that practically all marine life is naturally and inevitably richer in certain of the mineral elements than products from the land. The problem of the removal of arsenical spray residues from fruits and vegetables in order to make those foods safe for human consumption has recently focused the attention of investigators on the naturally high arsenic content of seafoods. Because of the increasing concern which is being shown by scientific workers at the present time the authors have undertaken to study the characteristics of the arsenic as it is contained in marine products when fed to laboratory animals.

Diets	Arsenic content of diets	Total ingested per rat (Milligrams arsenic as As_2O_3)		Total stored per rat (Milligrams arsenic as As_2O_3)		Per cent. of intake stored by rats	
	Mgm As_2O_3 per kilo	3 mo.	5½ mo.	3 mo.	5½ mo.	3 mo.	5½ mo.
Stock diet[1]	0.20	0.24	0.46	—	0.07	—	14.3
Stock diet + As_2O_3	17.90	19.78	36.32	3.73	3.58	18.8	9.9
High arsenic shrimp[2]	17.70	19.23	36.26	0.13	0.26	0.7	0.7
Low arsenic shrimp[2] + As_2O_3	17.90	19.60	36.27	3.57	4.25	18.2	11.7
Low arsenic shrimp[2]	1.20	1.29	2.49	0.11	0.18	8.1	7.1

[1] Sherman Diet 13 modified by Russell.
[2] The meat scraps (10 per cent.) of the stock diet replaced by dried shrimp.

There is no direct experimental evidence available at the present time which proves that the natural arsenic in marine foods, if consumed regularly over long periods of time, will or will not produce harmful physiological effects. However, the fact that seafoods are eaten regularly by maritime peoples and have for centuries constituted the principal article of diet of some of the nations of the world without demonstrable harmful effects is presumptive evidence that the arsenic present therein is in a relatively non-toxic form.

In the investigative work reported here shrimps were chosen as the source of "naturally combined" arsenic, since it is known that arsenic occurs in shrimp in greater concentrations than in most other seafoods. Samples of shrimp have been encountered which range in arsenic content (as As_2O_3) from 171 to 5 milligrams per kilogram, dry basis.

Groups of rats were taken at weaning and fed diets of various arsenic content derived from shrimp and from added arsenic trioxide. At the end of 3 months and again at 5½ months of the feeding period representative animals from each group were killed, autopsied and sections of their liver, spleen and kidney examined histologically for evidences of injury due to arsenic feeding. The carcasses of the animals (without the alimentary canal) were also analyzed for arsenic.

It can be seen from the results shown in the accompanying table that although the carcasses of rats which had received the largest amount of arsenic, in the form of shrimp, contained about 4 times the quantity contained in the stock diet controls (with no added arsenic), the rats which had received approximately the same quantity of arsenic as arsenic trioxide contained from 55 to 65 times that in the control animals. The results also show that during the first 3 months of the feeding period as well as for the full 5½ months only 0.7 per cent. of the ingested "shrimp arsenic" was stored in the bodies of the rats, while more than 18 per cent. of the inorganic arsenic trioxide was stored in the first 3 months. Apparently the rats receiving the inorganic arsenic trioxide had, some time within the first 3 months of the feeding period, reached an equilibrium in which no more storage of arsenic was taking place. It is, therefore, impossible to calculate from the above results the percentage of arsenic which was stored before this equilibrium had been reached. Undoubtedly the percentage stored would have been much higher had the first feeding period been of shorter duration.

The above results are direct evidence that there is a difference in the metabolism of the arsenic as it occurs in shrimp as compared to inorganic arsenic and that only a very small percentage of the arsenic contained in shrimp is absorbed and stored in the animal body when such foods are eaten.

There was no retardation of growth in any of the arsenic-fed animals nor any observable differences in their physical vigor or appearance and in none of them was there any histological evidence of injury to the spleen, liver or kidney due to the feeding of arsenic at the levels here employed.

These experiments are being continued, with other rats scheduled to be killed at the end of 9 and 12 months.

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EXPERIMENTAL PRODUCTION OF INCREASED INTRACRANIAL PRESSURE

THE production of a marked elevation in intraocular pressure by injecting small amounts of chloroform into the carotid artery of anesthetized dogs was described by Koppányi and Allen.[1]

We studied the effects of this procedure on the intracranial pressure. Dogs anesthetized with sodium barbital were used. Cisternal puncture was carried out and the needle connected with a water manometer. Blood pressure was recorded from one carotid artery, the other being used for chloroform injections. Injection of 0.2 to 0.5 cc of chloroform into the intact carotid artery produced a marked and sustained elevation of intracranial pressure. This elevation began in one or two minutes, reached a maximum in ten to twenty minutes, and remained approximately at the maximum level until the animal died. The usual level of the intracranial pressure before the chloroform injection was in the vicinity of 150 mm of water: the maximum height following chloroform injection was from 325 to 400 mm.

A coincident rise in intraocular pressure was produced by chloroform injections, corresponding to the observations made by Koppányi and Allen.

Once this elevated intracranial pressure was produced, the new high level seemed to be maintained much the same as the normal level. It varied directly with the level of arterial blood pressure. Alterations of arterial blood pressure resulting from the injection of epinephrine hydrochloride (marked rise), intravenous morphine sulfate (fall), intravenous 50 per cent. dextrose solution (initial fall and subsequent rise) and inhalation of amyl nitrite (marked fall) were invariably and immediately reflected by similar alterations in the increased intracranial pressure. Venous pres-

[1] *Proc. Soc. Exp. Biol. and Med.*, 12: 488, 1924–25.