these instances was always small—of the order of 5% in favour of the one or the other method. The average retention of the original vitamin C in all the experiments performed was 7.5% more in pressure cooking than in saucepan cooking—66% against 58.5%.

The estimations of vitamin C in the cooking-water showed that, with the exception of spinach, there was a greater loss by leaching in the saucepan than in the pressure pan. The results with spinach were no doubt due to the fact that no water was added to the saucepan, whereas 100 ml. was added in the pressure cooker. It is often recommended that the water used in cooking vegetables should be used in soups, stews, and gravies. This is, however, not often done, and when it is the liquid is frequently kept for some time and then cooked again. If it were to be used immediately the difference in the total ascorbic acid in the vegetable and cooking-water together would be negligible when comparing the two methods of cooking. In other words, the total amount of the vitamin in the vegetable and cooking-water together was on the average almost the same in the two forms of cooking (78% pressure cooking; 79.6% saucepan cooking).

Discussion

This investigation is of necessity limited by difficulties peculiar to any attempt to bring laboratory methods to domestic problems. On the one hand, it is clearly necessary to control as many factors as possible by using standardized conditions; on the other hand, it is obvious that no two housewives will cook in identical conditions; in fact, these conditions will differ from time to time even in one kitchen.

In the controlled conditions we have used it is evident that, so far as the retention of vitamin C is concerned, pressure cooking is certainly no worse than cooking in a saucepan, and in some instances is better. The differences are small, however, so that a bad technique with either method would result in a greater loss of the vitamin with that method. In some of our work, overcooking in the pressure cooker or a longer time in reaching the necessary pressure led to an appreciably greater loss of vitamin. Again, in what is still, unfortunately, the common way of cooking vegetables in a saucepan, with large volumes of water and a tendency to overcook, there is a greater loss of vitamin C than in the Ministry of Food method used in these experiments. It is probable, therefore, that either type of cooking in the home will often cause greater loss of vitamin C than in our experiments. This is more likely to occur when the saucepan is used: overcooking of vegetables in the pressure cooker results so easily in disintegration that housewives soon learn to avoid this, whereas slight overcooking with a large volume of water in the saucepan results in no obvious loss in appearance or palatability. We may reasonably conclude that, in general, the retention of vitamin C in vegetables in pressure cooking is likely to be no worse, and will probably be better, than in the saucepan when either process is used in the home. There is certainly no reason, from our experiments, to suppose that pressure cooking should be discouraged because of its effect upon the nutritive value of vegetables.

So far as other foods and vitamins are concerned, very little has been published. A few experiments of our own on the vitamin B₁ content of pulses suggest that the pressure cooker is better than the saucepan.

Summary

A comparison has been made of the effect upon the retention of vitamin C in 10 different vegetables when cooked in a modern pressure saucepan and in an ordinary saucepan according to the recommended methods. On the average the

retention was higher in vegetables cooked in the pressure cooker.

We should like to thank Professor John Yudkin for his counsel, and the Ministry of Food for cooking some of the samples.

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SPERMATOLYSIS: A CAUSE OF MALE STERILITY

BY

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Much attention has been directed of late towards the problem of sterility and infertility. In consequence I have been stimulated to present the results of some studies on male sterility which suggest a causative factor not hitherto described.

During the investigation of 75 males complaining of sterility at the urological clinic of Dr. A. E. Gussel, in the Guraba Hospital, Istanbul, I came across two specific abnormal spermatozoon types which were thought to be the cause of the sterility. The purpose of this communication is to call attention to the abnormalities. Short summaries of five of the case histories are given.

Case Reports

Case 1.—H. T., married five years; no children; wife pronounced normal by the gynaecologist. No history of venereal disease. No physical abnormality present. Examination of sperm 30 minutes after collection showed no microscopical or macroscopic abnormality. After two hours the specimen was separating into two layers—a clear supernatant and a sedimented layer. Microscopically the clear layer was acellular, but the sediment contained aggregations of dying spermatozoa. (I intend to make some observations on the death of spermatozoa in a further paper.) As time passed fewer and fewer sperms were seen, until at the end of six hours none were visible—the phenomenon of spermatolysis. Furthermore, the fluid obtained from this patient by prostatic massage caused spermatolysis to occur in the semen of a normal control when added to the latter.

Case 2.—N. O., married four times in the previous twenty years. One of his former wives had remarried and had had a family. No previous venereal disease, and no abnormal physical findings. The seminal fluid appeared to be more viscous than normal, but microscopically it seemed normal in every way up to two hours, when agglutination was beginning. After six hours spermatolysis was complete as in Case 1, and again prostatic fluid from this case caused spermatolysis when added to a normal control seminal fluid.

Case 3.—H. D., aged 34; married eight years; no history of venereal disease; wife normal. A spermogram showed oligospermia and hypokinesia. In two hours spermatolysis was complete.

In these three cases aspiration of the epididymis was performed. Unfortunately no sperms were found in any of the specimens, but in Case 3 minute comma-shaped bodies about 2 by 4 μ were seen. They showed some brownian movement. I got the impression that they were microspersms. Within two hours they had undergone lysis. In normal cases spermatozoa aspirated from the epididymis live two to three days.

Case 4.—F. O., aged 32; married three years; history of gonorrhoea twice, with clinical cure; no evidence of epididymitis or prostatitis; wife normal. Semen appeared normal

but showed asthenospermia microscopically. In five hours spermatolysis was complete, but in this case the prostatic fluid had no effect on a normal seminal fluid.

Case 5.—H. O., aged 30; married eight years. Nothing relevant in the history, and physical examination normal. Asthenospermia was present, and spermatolysis was complete in two hours.

Study of these cases suggests that two abnormal spermatozoon types were concerned. In the first type, as exemplified by Cases 3, 4, and 5, oligospermia and hypokinesia were present. Can it be assumed that the factors causing this also caused the spermatolysis? In the second type, as in Cases 1 and 2, the only abnormal feature was the spermatolysis. In all cases it seems right to assume that the spermatolysis was the cause of the sterility, since any sperm undergoing lysis within four to six hours of ejaculation is not likely to fertilize an ovum.

If spermatolysis occurs, then it seems reasonable to suggest that there must be a spermatozoon. In Cases 1 and 2 the only abnormality was spermatolysis, yet prostatic fluid from them caused lysis in the seminal fluid of normal controls. I have concluded that the prostatic secretion of these men contained a "spermatozoon" substance. The epididymis, vas deferens, seminal vesicle, and prostate produce a secretion which nourishes the sperms. Changes in the nature of the trophic products, the result of infection or from unknown causes, can give rise to abnormalities of the spermatozoa—e.g., asthenospermia—but I believe that the actual lysis factor is an abnormal prostatic substance.

Summary

Some cases of sterility in the male are due to agglutination and dissolution of the spermatozoa within a short time of ejaculation. This is spermatolysis.

Spermatolysis was thought to be the cause of sterility in five of 75 cases investigated. In two of these five no abnormality of the sperms apart from lysis was present.

The importance of examining seminal fluid at intervals is stressed. Otherwise if only one examination is done, and that too early, the phenomenon of spermatolysis might not be recognized.

Spermatozoon may be associated with otherwise normal spermatozoa or with abnormal sperm types. In the latter case it may be that a lysis factor is produced in the epididymis, vas deferens, or seminal vesicle, as well as in the prostate.

Medical Memorandum

A Case of Diabetic Coma Treated with 56,000 Units of Insulin

The following notes concern a case of diabetic coma treated at the General Hospital, Jersey. Their interest lies in the dose of insulin given and the conclusions they suggest concerning the use of glucose in diabetic coma.

CASE HISTORY

The patient, a man aged 32, a known diabetic, had previously kept well on a dose of 26 units of P-Z insulin and 16 units of soluble insulin. On Nov. 1, 1948, he had a cold; the following day he felt worse and omitted his insulin and took no food.

At 3 a.m. on Nov. 3 he was admitted to hospital in diabetic coma. Dehydration, air hunger, and the smell of acetone were conspicuous. The urine was "brick red" to Benedict's test and laden with acetone. The temperature was 98.6° F. (37° C.), pulse 102, and respirations 24. Gastric aspiration resulted in about 4 pints (2.28 litres) of dark-brown fluid. 130 units of insulin were given, and an intravenous drip of 5% glucose and 0.5% saline was set up. The blood sugar (B.S.) on admission was 500 mg. per 100 ml. Between 7 a.m. and 4 p.m. 950 units of insulin were given. At 6 p.m. the B.S. was 450 mg. per 100 ml. The pulse had risen to 130 and the patient's condition was worse.

In view of the inefficacy of the above method of treatment it was decided to change to that advocated by Professor Micks. As will be apparent, the inadequate treatment hitherto had permitted a marked state of insulin resistance to occur. At 7 p.m. the glucose drip was replaced with saline. A dose of 500 units of insulin was given and thrice repeated. The B.S. fell to 280 mg. 10,000 units were given at 1.30 a.m.: B.S. 270 mg. At 2.30 a.m. the B.S. had risen to 280 mg. and the patient had collapsed, the pulse being barely perceptible. At the suggestion of the laboratory technician the drip was investigated and was found to have been replenished in error with 5% glucose about an hour previously. The error was remedied and the patient improved.

At 3.15 a.m. 20,000 units of insulin, occupying 250 ml., were injected into several muscle masses, and 400 units into the drip tubing. At 4 a.m. the B.S. was still 280 mg. A second dose of 20,000 units was given and, since soluble insulin was running short, 400 units of P.Z.I. This proved to be the final dose. In 26 hours 56,080 units of insulin had been given.

Hitherto the urine (tested hourly) had been continuously "red" to Benedict's test and "acetone + + +." Now at last it began to become concentrated, and sugar and acetone to diminish. It did not become free from sugar and acetone till 22 hours after the last dose of insulin. The B.S. at 8.30 a.m., four hours after the last dose, was 65 mg., the pulse rate 160; the latter thereafter fell steadily and 24 hours later it was 90. No sudden change in the patient reflected the fall in blood sugar. Following the 8.30 a.m. B.S. result, glucose in amounts varying from 5 to 15 g. in 25% solution was administered hourly by drip tubing.

At 5 a.m. on Nov. 5, 25 hours after the last dose of insulin, a hypoglycaemic convulsion occurred without warning. It responded instantaneously to intravenous glucose. Shortly after this the drip was changed to glucose. By evening on the same day the patient was restored to consciousness sufficiently to take additional glucose by mouth. By about 11 p.m., some 65 hours after admission and 43 after the last dose of insulin, he was perfectly *compos mentis*. The drip was discontinued next day, and the patient was nursed flat for three days as a precaution against sudden circulatory failure. He progressed steadily and was discharged on Nov. 25, feeling perfectly fit, on a morning dose of 40 units of P.Z. and 60 units of soluble insulin.

COMMENT

It is to be supposed that the patient's descent into coma was initiated by the cold he had on Nov. 1. No trace of this or evidence of any other infection was apparent on admission. I had expected the conversion of the B.S. from high to low values to be indicated by marked clinical change. A sharp rise in the pulse rate coincided with the event, but this was the only prominent point. The pulse rate and the urinary sugar and acetone gradually diminished, and the patient's general condition slowly improved. Even the hypoglycaemic convulsion, arriving late and without warning and immediately controlled, did not seem to interrupt this steady progress.

B.S. recordings during this period may be of interest. These were: 5 hours after the last dose of insulin, 65 mg. per 100 ml.; 7 hours after last dose, 56 mg.; 8 hours, 50 mg. (here the drip was changed to glucose); 10 hours, 62 mg.; 12 hours, 30 mg.; 14 hours, 50 mg.; 16 hours, 24 mg. (no clinical reflection of this low value); 27 hours, 70 mg. (convulsion two hours before this); 29 hours, 60 mg.; 32 hours, 45 mg.; 34 hours, 168 mg.

The lack of response to treatment when glucose was used, the collapse that occurred when it was added in error to the saline drip, and the recuperation that followed the correction of the error, strongly suggest that glucose is not an innocuous substance in diabetic coma and that its use as a precaution against hypoglycaemia may gravely jeopardize the patient's chances of survival. This was emphasized by Root (1945) and Micks (1948).

Gastric aspiration on admission produced brown fluid containing altered blood. A second aspiration twelve hours later yielded about 1 oz. (28 ml.) of blackish fluid, presumably also blood further altered. This, I believe, is a usual occurrence in diabetic coma. I should be interested to know the cause and origin of this blood. I understand that a surgeon operating on a case of chronic pyloric stenosis may occasionally observe blood oozing from the gastric mucous membrane.

I wish to thank Dr. Graeme Bentlif, senior physician to the General Hospital, for permission to publish these notes.

J. G. H. SHEPPARD, M.B., B.S.

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