muscle stump showed good movement and the final prosthetic motility was satisfactory. The average motion obtained with the prosthesis when the ball and ring implant was used, is shown in Tables I and II.

TABLE I.

PROSTHETIC MOVEMENT OBTAINED USING BALL AND RING AT THE TIME OF ENUCLEATION

Direction of gaze	*Average degrees of movement*
Nasally	33
Superiorly	33
Temporally	32
Inferiorly	27

TABLE II.

PROSTHETIC MOVEMENT OBTAINED USING BALL AND RING IN SOCKETS SOME TIME AFTER ENUCLEATION

Direction of gaze	*Average degrees of movement*
Superiorly	29
Nasally	28
Inferiorly	28
Temporally	23

Complications.—One patient developed orbital cellulitis nine months after operation. She was hospitalized for ten days and the swelling subsided under intramuscular penicillin treatment. In another patient the socket was scarred following removal of the eye two years previously because of a hand grenade explosion. Two weeks after the implant was inserted, conjunctiva and Tenon's capsule retracted over the upper part of the ring. Later attempts to replace these tissues were unsuccessful; however, the implant has not extruded and the prosthesis still shows good movement.

CONCLUSIONS

1. The basket implant operation is superior to older methods. The prosthetic movement is more spontaneous and of greater range than that obtained with no implant, or with a ball implant. Later complications are unlikely because the implant is covered with Tenon's capsule and conjunctiva.

2. In contrast to use of basket in original cases, the results obtained when the basket implant is used in sockets where the eye had been removed some time before is not satisfactory. It is not necessary to have a custom-made eye with a stud, as good movement can be obtained with a stock eye, and if convenient, a stud can be fitted later.

3. The range and spontaneity of movement of the prosthesis with the ball and ring implant is even better than with the basket type. Best results are obtained when this implant is used at the time of the original enucleation, but satisfactory results can be obtained when used in sockets where the eye had been removed some years before.

SUMMARY

Two newer types of implants used after enucleation have been described—namely, the basket and ball and ring. The results using these implants in 36 cases are given. The surgical technique using ball and ring implant is outlined.

The author expresses his appreciation to Dr. A. Lloyd Morgan for many helpful suggestions regarding surgical technique.

The above series includes several cases from the practices of Dr. A. Lloyd Morgan, Dr. A. J. Elliot, and Dr. W. R. F. Luke, with their kind permission.

The implants and artificial eyes were made at Brent Laboratories, 62 Avenue Road, Toronto.

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A NEW PARASYMPATHETIC STIMULANT —ETHYL 3:3 DIMETHYLALLYL BARBITURIC ACID*

1. Its Effect on Gastric Secretion

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DURING the war extensive investigations were conducted on the use of barbiturates to control motion sickness.[1] It was found that certain derivatives exerted a protective action presumably by a central effect and that this was not necessarily related to the hypnotic or anæsthetic power of the barbiturate.[2] Since the object of these experiments was to find if such a class of compounds might affect other actions of the nervous system a number of tests were made on gastric function. As has been pre-

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Supported in part by a grant from the Bristol-Myers Company.

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viously reported by one of us (R.L.N.) two barbiturates ethyl 3:3 dimethylallyl barbituric acid (No. 16-A*) and ethyl 1:3 dimethyl-1-butenyl barbituric acid (No. 21-A) were found to be powerful stimulants of gastric secretion in cats.[3] Another compound (1-methylbutyl) ethyl acetyl thiourea (24-A) was found to inhibit the gastric secretion which normally follows the central stimulation caused by the hypoglycæmic action of insulin.[4] This substance also inhibited the stimulating action of No. 16-A. Although it was suggested that the effects of these compounds could be explained by a central action it was essential to continue and extend these observations. The present paper concerns further studies on the effect on dogs of compound 16-A.

Methods.—Twelve dogs with gastric fistula were used, three having their vagi destroyed previous to use of drug. In two, vagotomy was performed after control experiments. Except where otherwise stated the dogs were unanæsthetized, and fasted for approximately 24 hours. Every possible effort was made to prevent any psychic secretion, by first of all the experimenter never feeding dogs, and secondly tests being carried out in a separate room, where the dogs were never fed or handled. The stomach was washed with water, and drained thoroughly before beginning the experiment. Gastric secretion was collected continuously, with control periods of varying intervals to ensure that the gastric glands were quiescent. The drug was dissolved in water and injected subcutaneously. The volume of gastric juice was measured and the pH estimated by using a Beckman pH meter, and pepsins were estimated by Le Veen[5] method. When the blood sugar levels were calculated the Folin-Wu[6] method as adapted to Evelyn photocolorimeter was used. Although in these experiments the drug was always used subcutaneously it is also effective orally.

Results.—The results of this investigation are reported under the following headings: (1) Effect on gastric secretion. (2) Site of action of drug. (3) Other actions of 16-A.

1. *Effect of 16-A on gastric secretion in normal dogs.*—The dosage of the drug used varied usually between 3 and 4 mgm./kg. subcutaneously. To demonstrate this effect 9 normal fistula dogs were used, and 20 administrations of the drug given. The following are examples of results obtained:

Dog No. 26, 36 kg. 16-A 4 mgm./kg. subcutaneously at 1045 hrs.

GASTRIC SECRETION

Time	*Vol.*	*pH*	*Pepsin units/c.c.*
0930 - 1000	6.0 ml.	1.79	1,803
1000 - 1030	9.0 ml.	4.10	
1030 - 1045	3.0 ml.	4.10	
1045 - 1115	13.0 ml.	3.87	
1115 - 1145	205.0 ml.	1.27	4,633
1145 - 1215	257.0 ml.	1.40	8,625
1215 - 1245	175.0 ml.	1.45	88,318
1245 - 1315	92.0 ml.	1.24	
1315 - 1345	45.0 ml.	1.08	279,188

Dog No. 33, 24.5 kg. 16-A 4 mgm./kg. subcutaneously at 0950 hrs.

GASTRIC SECRETION

Time	*Vol.*	*pH*
0830 - 0930	1.0 ml.	neutral
0930 - 1000	10.0 ml.	5.95
1000 - 1030	76.0 ml.	1.80
1030 - 1100	66.0 ml.	1.52
1100 - 1130	70.0 ml.	1.81
1130 - 1200	22.0 ml.	1.47

The above results on pepsin are representative of several experiments since as the secretion becomes less, pepsin values rise. Whether this is due to a dilution or a psychic factor is not clear.

This drug is a very potent stimulus of gastric secretion. An idea of just how potent may be obtained by comparing results of 16-A and insulin induced secretion.

Dog No. 28, 24.5 kg. Insulin induced secretion. Fasting gastric secretion collected for ½ hr. control period, then 8 units insulin given intravenously at 0945 hrs.

GASTRIC SECRETION			BLOOD SUGARS	
Time	*Vol.*	*pH*	*Time*	*Mgm. %*
0915 - 0945	.... 1.0 ml.	3.98	0940	82.5
0945 - 1015	.... 10.0 ml.	3.48	1015	41.0
1015 - 1045	.... 27.0 ml.	1.07	1045	47.0
1045 - 1115	.... 12.0 ml.	1.20	1115	64.0
1115 - 1130	.... 4.0 ml.	1.11	1130	65.0

In all these experiments, visible mucus was present in every sample. For results with 16-A see Dog No. 28, Section 2.

2. *Site of action of drug.*—(*a*) In view of the similarity between the effect of this drug and that of insulin hypoglycæmia, on gastric secretion, it was conceivable that it might cause its effect by lowering of blood sugar. This however was found not to be the case as is shown by the following experiments.

* Compounds Nos. 16-A, 21-A, and 24-A were prepared and kindly supplied by the Lilly Research Laboratories, Eli Lilly & Company.

Dog No. 40, 30 kg. Fasting gastric secretion collected for 1 hr. Fasting blood sugar collected at 1015. Then 16-A 3 mgm./kg. subcutaneously at 1020 hrs., and blood sugars estimated at ½ hr. intervals.

Gastric Secretion			Blood Sugars	
Time	*Vol.*	*pH*	*Time*	*Mgm. %*
0930 - 1000	.... 4.0 ml.	4.30		
1000 - 1020	.... 8.0 ml.	7.60	1015	85.5
1020 - 1050	.... 12.0 ml.	7.40	1050	78.5
1050 - 1120	.... 53.0 ml.	1.75	1120	85.0
1120 - 1150	.... 73.0 ml.	1.38		
1150 - 1220	.... 64.0 ml.	1.34		

Dog No. 28, 24.5 kg. Fasting gastric secretion collected for 1 hr. Fasting blood sugar collected at 0920 hrs. Then 16-A 3 mgm./kg. injected subcutaneously at 1000 hrs. Blood sugar estimated at ½-hr. intervals.

Gastric Secretion			Blood Sugars	
			Time	*Mgm. %*
0900 - 0930	... 35.0 ml.	Acid		
0930 - 1000	... 6.0 ml.	"	0920	93.0
1000 - 1030	... 22.0 ml.	"	1030	115.0
1030 - 1100	... 160.0 ml.	"	1100	84.0
1100 - 1130	... 145.0 ml.	"	1130	115.0
1130 - 1200	... 55.0 ml.	"		

(*b*) *Effect on gastric secretion following vagotomy*:

Dog No. 39, 23.6 kg. Experiment previous to operation. Fasting gastric secretion collected for 1 hr. period. Then 16-A 3 mgm./kg. injected subcutaneously at 1020 hrs.

Gastric Secretion		
Time	*Vol.*	*pH*
0920 - 1020	5.0 ml.	2.16
1020 - 1050	2.0 ml.	6.97
1050 - 1120	39.0 ml.	1.42
1120 - 1150	62.0 ml.	1.11
1150 - 1220	51.0 ml.	1.10
1220 - 1250	40.0 ml.	1.00
1250 - 1320	40.0 ml.	1.02
1320 - 1350	63.0 ml.	0.98

Dog No. 39, 24 kg. Supradiaphragmatic vagotomy done, and after a satisfactory preoperative period, 16-A 3 mgm./kg. injected subcutaneously at 1030 hrs.

Gastric Secretion		
Time	*Vol.*	*pH*
1000 - 1030	2.0	4.23
1030 - 1100	2.0	4.18
1100 - 1130	2.0	alkaline
1130 - 1200	2.0	7.82
1200 - 1230	2.0	alkaline
1230 - 1300	2.0	neutral

In four more dogs, similar results were obtained in 6 experiments. The vagi in these experiments had been destroyed by electrodes. This nerve destruction was confirmed by extreme gastric retention present 24 hours after feeding, and no gastric secretion following insulin hypoglycæmia. In these cases an insulin test was not considered satisfactory unless the blood sugar fell to below 50 mgm. %.

2. (*c*) *Prevention of 16-A induced gastric secretion by use of tetra ethyl ammonium chloride* ("Etamon", Parke-Davis & Co.).—Insulin induced gastric secretion can be prevented by etamon. This blockage appears to take place at the junction of pre- and post-ganglionic neurons.[7] For control experiment without etamon, see Dog No. 33, Section 1.

Dog No. 33, 24.5 kg. Dog fasting only 16 hrs. and intestinal phase of gastric secretion still occurring. Gastric secretion collected for ½-hr. period then 16-A 3 mgm./kg. given subcutaneously at 0930 hrs. Etamon 10 mgm./kg. intravenously at 0950 hrs.

Gastric Secretion		
Time	*Vol.*	*pH*
0900 - 0930	18.0 ml.	1.39
0930 - 1000	2.5 ml.	1.52
1000 - 1030	2.0 ml.	5.41
1030 - 1100	4.0 ml.	7.91
1100 - 1205	13.0 ml.	8.23

2. (*d*) *Prevention of 16-A induced gastric secretion by nembutal anæsthesia.*—Three such experiments were carried out on different dogs. The following is an example of such an experiment:

Dog No. 40, 30 kg. For control experiment with 16-A only, see under section (2*a*). Fasting gastric secretion collected for ½-hr. period. Nembutal 25 mgm./kg. intravenously at 0930 hrs. Then 16-A 3 mgm./kg. subcutaneously at 1000 hrs.

Gastric Secretion		
Time	*Vol.*	*Acidity*
0900 - 0930	5.0 ml.	weakly acid
0930 - 1000	0.0 ml.	
1000 - 1030	0.0 ml.	
1030 - 1100	0.0 ml.	
1100 - 1130	0.0 ml.	
1130 - 1200	0.0 ml.	

To summarize.—Gastric secretion induced by 16-A does not result from lowering of the blood sugar; vagotomy prevents this secretion; blockage of parasympathetic by etamon prevents secretion as does nembutal anæsthesia. These facts all show that 16-A activates gastric secretion via the parasympathetic nervous system. It appears to act centrally, further observations seem to place this effect on the hypothalamus.

3. *Other effects of 16-A.*—In addition to stimulating gastric secretion marked stimulation of the salivary glands occurs. So marked is this secretion that the dogs drool saliva from the mouth, swallow frequently, and in addition to these findings it has been noted that 3 mgm./kg. causes a prominence of the nictitating membranes, paralysis of the pupil in semidilatation, the pupils not reacting to light. If 4 mgm./kg. are given this paralysis of the nictating mem-

branes becomes more marked so that nearly half the eye is covered, both eyelids are weakened, and endopthalamus develops. This picture usually begins about 20 minutes after injection, approximately ten minutes before gastric secretion commences, and usually lasts for one hour. These findings would suggest a sympathetic paralysis.

This drug also appears to cause symptoms of stimulation and in doses of 4 mgm./kg. the dogs are quite restless, whining, and moving in their stands. Involuntary micturition and defæcation sometimes occur. In addition to the above effect, 5 mgm./kg. usually results in marked excitement, continuous barking, and biting at any object within reach, weakness is very marked and if dogs are not restrained they will run about wildly, with a marked staggering gait.

SUMMARY

1. Sodium ethyl 3:3 dimethylallyl barbituric acid is a powerful stimulant to gastric secretion. It strongly stimulates acid and mucus cells but apparently acts as only a weak stimulant to peptic cells.

2. Its action is central, and is prevented by vagotomy, etamon, and nembutal anæsthesia.

3. There is also an apparent paralysis of the nictitating membrane and reaction of the pupil suggesting a depression of sympathetic function. Larger doses are followed by symptoms of excitement, and inco-ordination.

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In India, a million acres of safflower are grown each year. The thistle-like flower is used to make a yellow dye, the leaves are used in salads, and oil from the seeds as food and in paint.

THE NEUROGENIC ORIGIN OF BLADDER SYMPTOMS FOLLOWING PROCTECTOMY*

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OF recent years clinicians have become increasingly aware of bladder dysfunction following radical resection of the rectum. Whipple[1] reported in 1932 that half of his cases of abdomino-perineal operations were followed by cystitis, but he did not mention any urinary functional impairment. Hill, Barnes and Courville[2] by means of cystograms produced excellent evidence that the postoperative difficulties were neurogenic in origin. Their findings showed that the bladder was passively distended owing to atonicity of its musculature. An interesting cystogram in one case revealed that the trauma to the nerve fibres to the bladder was more severe on one side than on the other, as evidenced by the greater prominence of one side of the vesical dome.

On the other hand, Coller and Eastman[3] state that "abdominal-perineal resection of the rectum destroys neither the autonomic nor the somatic nerve supply to the bladder". They based their conclusions on a comparison of pre- and post-operative cystometrograms and believed that the urinary retention after this operation was probably due to "local trauma and reflex inhibition".

The diversity of opinion regarding these urinary complaints shows that the causative mechanism is by no means clear. No one explanation has been generally accepted. Part of the reason for this persisting problem must undoubtedly be that bladder neurophysiology and the mechanism of micturition are not well understood.

Both during and following the war, one of us (C.A.) has been responsible for the genito-urinary care of 153 paraplegics. A considerable number of these cases have required trans-urethral resections of their bladder necks for vesical urinary retention, and as a consequence we have become familiar with the cystoscopic appearance of the bladder neck and posterior urethra in paraplegic patients. Because of this added experience it was felt that cystoscopic

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