3. The selection of cases, statistical analysis, sputum bacteriology, endobronchial disease, treatment before resection, type of lesions, indication and type of resection, surgical mortality and complications are discussed.

4. In this series there has been but a single death and this was due to pulmonary embolism. The operative death rate is 0.7%. No late deaths have been reported with a follow-up from three to 24 months. The total complication rate has been 8.1%.

5. The present position of the patients is reviewed.

6. The surgical points which in the author's experience were basic in securing the results are discussed.

It is a pleasure to acknowledge the valuable assistance of Dr. J. Orr, Director of the Medical Services the Saskatchewan Anti-Tuberculosis League, in the preparation of this study and in giving permission to publish it. Grateful thanks are due to the superintendents, surgical assistants, and medical, laboratory and x-ray staff of the three sanatoria for the full co-operation we have been given throughout.

We are also indebted to Dr. J. W. Adams, pathologist of the City Hospital, for pathological examination and report on all our resected specimens.

ADDENDUM: At the time of publication of this paper 168 consecutive resections have been performed without fatality.

NOTE—A comprehensive list of 55 references has been prepared and may be obtained from the author on request.

Saskatoon Sanatorium.

RÉSUMÉ

1. La thérapie chimio-antibiotique a révolutionée les anciennes conceptions de la pathologie, de la bactériologie et du traitement de la tuberculose pulmonaire. Dans la majorité des cas la chimiothérapie de longue durée conduit la maladie jusqu'au stage "d'arrêt" ou "d'inactivité". Les bacilles tuberculeux prélevés des spécimens opératoires démontrent une incertain capacité de se réproduire dans les cultures et d'infecter les cobayes inoculés. On se demand si la résection est nécessaire et quels en sont les risques.

2. On rapporte une série personnelle de 135 résections pulmonaires pour tuberculose, 26 pneumonectomies, 61 lobectomies et 48 résections segmentaires en les analysant selon la bactériologie du crachat, le caractère des lésions, le traitement avant la résection, l'indication et le type de résection, la mortalité et les complications opératoires.

3. Dans cette série la mortalité opératoire a été de 0.7%, le seul décès ayant été causé par une embolie pulmonaire. On ne déplore aucun cas de décès tardif, après un contrôle clinique de 3 jusqu'à 22 mois. Le pourcentage des complications a été de 8.1%.

4. On décrit la condition présente des malades et on discute certains points de téchnique chirurgicale que l'auteur considère comme capitaux. F.F.A.

AN OUTBREAK OF PARALYTIC SHELLFISH POISONING

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PARALYTIC SHELLFISH POISONING was first recognized as a public health problem on the Atlantic Coast of Canada in 1936, when Murphy[1] reported that five cases with two deaths had occurred in Nova Scotia following the consumption of mussels. Other investigations revealed a serious problem,[2] and since 1943 collaborative studies have been conducted without interruption by the Department of National Health and Welfare, the Department of Fisheries, and the Fisheries Research Board of Canada. In 1947, Medcof *et al.*[3] presented a comprehensive report of an epidemiological study in which the toxicity of raw shellfish, the dosage of poison, and the resulting symptoms were correlated. They showed that the mildest symptoms of poisoning were observed only when a quantity of poison in excess of 1,000 mouse units was ingested, thus confirming the 400 mouse unit quarantine level first suggested by the work of Sommer and Meyer.[4] The clinical picture in the 28 human cases in New Brunswick in 1945 was carefully studied. The symptoms varied in severity with the amount of poison ingested, but were consistent: numbness about face and mouth, with paræsthesiæ of the lips; vomiting; headache; dizziness; difficulty in breathing; general weakness; occasional paralysis. Poisoning of domestic animals, particularly house cats, seems to have been common. A traditional knowledge of the dangers involved, and a degree of resistance to the poison, apparently have prevented more frequent illness among the inhab-

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itants of shore communities; many instances of shellfish poisoning have been reported among tourists and other visitors to the fishing areas for whom shellfish were not a habitual item of diet.

Medcof *et al.* concluded that the minimum amounts of poison required to produce mild, severe, and extreme symptoms of poisoning in susceptible persons might be in the neighbourhood of 2,000, 10,000 and 25,000 mouse units respectively. Domestic cooking processes, including steaming and pan-frying, were found to reduce the poison content of raw clam meats by at least 70%, but failed to provide sufficient protection in many cases.

Gonyaulax tamarensis, a species of dinoflagellate on which shellfish feed, has been shown to be the ultimate source of the poison.[3] A close correlation between large numbers of the dinoflagellate in seawater and the appearance of high toxicities in shellfish has been observed, usually between mid-July and the end of September on the Canadian Atlantic coast. All of the molluscan shellfish found in the Bay of Fundy have been shown to be toxic to some degree at such times; the soft-shell clam (*Mya arenaria*) is the most important commercial species and, fortunately, is the least dangerous (oysters are not found in the Fundy area, and as a species have never been shown to be toxic). Since 1943 the Department of Fisheries, on recommendation from the Department of National Health and Welfare, has imposed temporary restrictions on the taking of shellfish from toxic areas in New Brunswick; at such times the clam beds have been closed to all fishing except for canning. All canned packs from toxic areas are routinely checked for toxicity before release. Dangerous areas have been posted with warning signs. There has been no difficulty in maintaining a reliable control over all commercial operations, but it has not been easy to enforce the fishing prohibition on the general public.

A shellfish toxicity problem has also been known to exist on the south shore of the St. Lawrence River since 1948, when two children died from eating what are believed to have been mussels (*Mytilus edulis*) collected at Les Boules, Que.[5] The recent discovery of large stocks of soft shell clams on the north shore of the St. Lawrence has stimulated interest in the toxicity problem and its effect on possible commercial exploitation of the clams. Since 1953 the Department of Fisheries of the Province of Quebec, in close co-operation with the Department of National Health and Welfare, has adopted control measures virtually the same as those employed in the regulation of the clam fishing industry in New Brunswick and Nova Scotia. Extracts from clam specimens taken periodically from producing areas on both shores of the St. Lawrence River are tested for toxicity at the Laboratory of Hygiene, Ottawa; when toxicity values reach the quarantine level of 400 mouse units, the areas are closed to commercial fishing and warning signs are posted on the beaches. Nearly 550 specimens of Quebec clams have been tested for toxicity since March 1953.

On July 6, 1954, a routine toxicity test indicated that soft-shell clams from Metis Beach on the south shore of the St. Lawrence River contained 419 mouse units of toxin, and the area was closed to fishing. A family of seven persons (the parents, Mr. and Mrs. A.G., a daughter-in-law, Mrs. R.G., a son, R.G., and three daughters, Mrs. R., Miss C.G., and Miss D.G.), visited Metis Beach, and on July 13 collected mussels (*Mytilus edulis*) from the beach. C.G. ate two or three mussels without ill effect; R.G. consumed an undetermined number of mussels and suffered from "upset digestion," while his wife, Mrs. R.G., who ingested a somewhat larger number of mussels, complained of stomach cramps and violent diarrhœa. The family cat ate an undetermined number of mussels, with subsequent paralysis and disturbed head movements. On July 14 all members of the family except Mrs. R. ate an indeterminate number of mussels collected at St. Flavie, P.Q., without ill effect.

On July 15, between 9:00 and 10:00 a.m., soft-shell clams were collected from the Metis Beach tidal flats. Although the G. family was informed of the danger from shellfish poison by residents of the area and by warning signs on the beach, the clams were steamed and eaten between 11:00 a.m. and 12:00 noon. No clam bouillon was ingested. Symptoms appeared within one-half hour after ingestion of the clams, and were the same in all members of the family (see Table I). The family cat refused to eat the clams and was not affected. All patients except Mrs. R. were induced to vomit within one hour after ingestion of the clams. Mrs. R. died at 12:45 p.m. and Mr. R.G. at 3:00 p.m. July 15. The others recovered by July 17, and were discharged from Rimouski Hospital on July 22.

TABLE I.

	Symptoms						
	Mr. R.G.	*Mrs. R.*	*Mr. A.G.*	*Mrs. A.G.*	*Mrs. R.G.*	*Miss C.G.*	*Miss D.G.*
Age	36		69	60	34	27	12
Clams ingested	32–40	12–15	12–15	5–6	5–6	3–4	3–4
Minimum weight (gm.)	326.7	122.5	122.5	51.1	51.1	30.6	30.6
Maximum weight (gm.)	408.4	153.2	153.2	61.3	61.3	40.8	40.8
Paræsthesiæ (lips)	yes	yes	yes	yes	yes	yes	yes
Occipital headache			yes			yes	
Vomiting	yes	nausea	yes	yes	yes	yes	yes
Paralysis	yes	yes	yes	yes	yes	yes	yes
Apathy			yes	yes	yes	yes	yes

Field Investigation

Two of the authors (J.N. and H.E.C.) established a sampling station at the point on the Metis Beach tidal flats where clams had been collected by the G. family; specimens of *Mya arenaria* were collected daily from this point from July 18 to July 22. Extracts were prepared as follows: the clams were shucked, washed in fresh water, drained on a sieve for 5 minutes, and thoroughly minced; a 100-gram portion of the minced meats was suspended in 100 ml. of 0.1 N hydrochloric acid, and boiled gently for 5 minutes with continuous stirring; the mixture was then cooled, made up to its original volume (200 ml. approximately) with distilled water, and the pH adjusted to between 4.0 and 4.5 by addition of a few drops of 5N acid or 0.1 N sodium hydroxide, using B.D.H. universal indicator internally. The supernatant liquid was clarified by settling, and constituted the extract which was shipped to the Laboratory of Hygiene for assay.

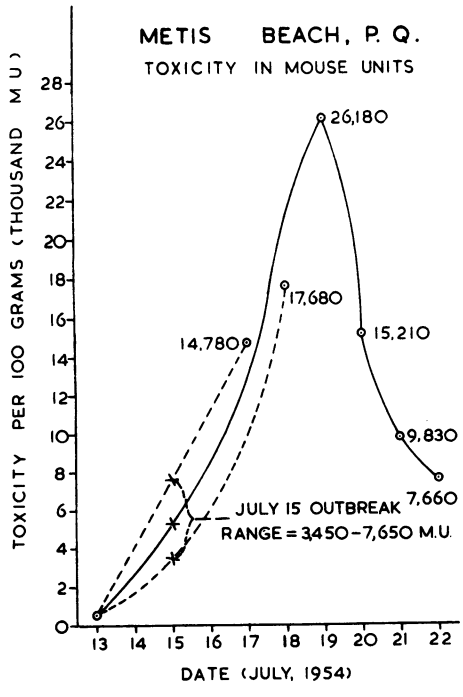

Fig. 1

One millilitre of the extract (and decimal dilutions) was injected intraperitoneally into each of three white mice (18 to 22 gm.), and their death times were measured to the nearest five seconds. The mean death time was referred to a standard toxicity graph from which toxicity was determined and expressed as "mouse units." All toxicities for *Mya arenaria* taken from Metis Beach during the study period are tabulated below, and are plotted on a graph (Fig. 1).

TABLE II.

Date	*Toxicity*	*Date*	*Toxicity*
July 13	542	July 20	15,210
July 17	14,780	July 21	9,830
July 18	17,680	July 22	7,660
July 19	26,180		

Replicate clam specimens from Metis Beach were shucked and weighed; the mean weight per clam was 10.21 gm.

Since no specimens were obtained from Metis Beach on July 14, 15 and 16, only a rough approximation of the probable toxicity level on July 15 can be obtained by interpolation on the graph. It is probable, however, that the clams eaten by the G. family contained from 3,450 to 7,650 mouse units per 100 gm. of meat.

Medcof *et al.*[3] concluded that steaming reduces the poison content of clam meats by at least 70%. Dosages were therefore calculated on the basis of the approximate level of toxicity in Metis Beach clams on July 15, and on the assumption that 30% of the poison remained after steaming. Dosages were also calculated for 100% retention of the toxin during steaming, to provide an estimate of the maximum possible dosage.

Conclusions

Because insufficient information is available, no very precise measure can be made of the dosages of shellfish poison which produced illness and two deaths in the seven members of one family. It is impossible to determine the exact poison content of the ingested clams, and the precise

effect of steaming on their toxin content. It is evident, however, that one death (R.G.) resulted from the ingestion of not less than 3,300 mouse units, and not more than 31,300 m.u.; if the loss of toxin due to steaming was approximately 70%, it is probable that approximately 5,800 m.u. of shellfish poison were ingested by R.G.

The second fatality (Mrs. R.) resulted from the ingestion of not less than 1,200 m.u., and not more than 11,700 m.u.; a dosage of approximately 2,400 m.u. is considered to be probable. A.G. survived a similar dosage; it is probable, therefore, that the dosage in Mrs. R's case approached the minimum lethal dose.

Mrs. A.G. and Mrs. R.G. ingested not less than 528 m.u. and not more than 4,700 m.u. (probably about 1,000 m.u.). Misses C.G. and D.G. ingested not less than 300 units and not more than 3,200 m.u. (probably about 650 m.u.).

The unfortunate outbreak of paralytic shellfish poisoning reported herein points up the need for continued rigid control of clam-producing areas. Clams with toxicities of 400 mouse units or more per 100 gm. of meat must be excluded from the market at all times.

The persons involved in the Metis Beach outbreak collected and ate the toxic clams in spite of what should have been adequate warning from local residents and from signs posted on the beaches. It may be necessary to take further measures for the protection of tourists unfamiliar with the shellfish toxicity problem.

REFERENCES

1. MURPHY, A. L.: *Canad. M. A. J.*, 35: 418, 1936.
2. GIBBARD, J., COLLIER, F. C. AND WHYTE, E. F.: *Canad. Pub. Health J.*, 30: 193, 1939.
3. MEDCOF, J. C. *et al.*: Paralytic Shellfish Poisoning on the Canadian Atlantic Coast, Bull. 75, Fisheries Research Board of Canada, 1947.
4. SOMMER, H. AND MEYER, K. F.: *Arch. Path.*, 24: 560, 1937.
5. MEDCOF, J. C.: Toxicity records, 1948. Atlantic Biological Station. Orig. MS Rep. 757 (unpublished), 1949.

THE PRACTICAL TESTING OF VESTIBULAR LESIONS*

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IN ORDER to determine the origin of vertigo it is essential, in addition to taking an accurate history, to have reliable test procedures. If the vertigo is due to an end-organ lesion, the patient recognizes at once the similarity of his attacks and the rotational vertigo artificially produced by clinical testing. Furthermore, by the use of unilateral stimulation each organ of balance can be tested separately, thereby providing significant information as to the side and the site of the lesion. The prompt recognition of this type of vertigo produced by peripheral stimulation is most significant since it differs from that due to central irritation.

Following the testing of over 200 consecutive patients referred for investigation of vertigo at St. Michael's Hospital, it appears evident that certain vestibular tests are of particular value. It is the purpose of this paper to suggest a routine procedure which has been found to be most practical.

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PROCEDURES

One of the most reliable of tests that can be carried out in the doctor's office is the *caloric test.* By raising or lowering the temperature of the petrous bone with the application of heat or cold to the ear drum, the fluid in the adjacent lateral semi-circular canal is induced to flow by convection currents. One simple method is to introduce 5 c.c. of ice water into the ear canal by means of a 10 c.c. syringe and needle of moderately small calibre, with the patient sitting up or in the prone position. The direction (quick phase) and duration of the nystagmus produced are then recorded. Normally with cold water (lower than body temperature) the quick phase beats to the opposite side; this should be reversed by warm water. After a few trials on normal patients, the physician can evaluate the hyperactive or hypoactive end-organ responses. Nystagmus produced in this fashion normally lasts two minutes. This procedure is highly recommended by McNally.[1] The interpretation of these responses will be discussed later.