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RÉSUMÉ

Les recherches continuent dans le domaine de l'épuration extra-rénale. Un groupe de la Colombie canadienne fait part de son expérience clinique dans l'emploi d'un rein artificiel de modèle récent (Kolff 1956) chez cinq malades accusant de l'oligo-anurie et souffrant d'une forme quelconque d'insuffisance rénale. Le principal objectif de ce traitement est de diminuer l'accumulation des déchets azotés et d'abaisser le taux de potassium dans l'organisme dans l'espoir d'un rétablissement éventuel de la fonction rénale. L'appareil consiste en deux serpentins jumelés de cellophane de neuf mètres chacun, fournis tout stérilisés par le fabricant et ne devant servir qu'une seule fois. Ces tubulures contiennent de 800 à 1100 ml. de sang sous pression dialysé contre 100 litres de solution préparée à cet effet. Le circuit abrégé de cet appareil provoque une surcharge hémodynamique moindre que celle que causait les modèles antérieurs, diminuant ainsi le risque d'œdème pulmonaire. La masse circulante de sang est moins forte et requiert une quantité d'anticoagulant moins élevée. La dialyse s'obtient plus rapidement puisqu'elle ne dépend plus uniquement de l'osmolarité; il est donc relativement aisé de rétablir l'équilibre hydrique. Enfin, l'appareil peut être préparé à moins d'une heure d'avis, ce qui n'est pas un de ses moindres avantages.

HISTOPLASMOSIS IN SOUTHWESTERN ONTARIO*

R. A. HAGGAR, M.D., M.Sc.,†
E. L. BROWN, M.D. and
N. J. TOPLACK, M.D., M.Sc.,
St. Thomas, Ont.

HISTOPLASMOSIS is the most common systemic fungus infection of man.[1] Once believed to be an invariably fatal disease, it has now been well established that the vast majority of cases are benign asymptomatic infections which are clinically unrecognized.[2, 3] Although the lungs are most frequently involved, the disease may be disseminated and affect almost every organ of the body. Excellent reviews of all aspects of the disease have been recently written by Peterson and Christie[1] and by Loosli.[4]

HISTORICAL SUMMARY

Histoplasmosis was first described in 1905 by Samuel Darling[5] in Panama who was studying the spleen, liver and bone marrow of all cases of splenomegaly autopsied at the Ancon Hospital in the Canal Zone. A few years earlier the Leishman-Donovan bodies of kala-azar had been described and Darling was searching for cases of this disease. He found no evidence of kala-azar in Panama but instead encountered three cases within a period of nine months which were characterized by the presence of cytoplasmic inclusions in the reticulo-endothelial cells of various organs. These inclusions appeared to be small, ovoid, encapsulated micro-organisms, 2 to 4 microns in diameter, and Darling, believing that they were protozoa, named them *Histoplasma capsulata*. He was unable to grow them on artificial media, and guinea-pig inoculations were unsuccessful.

Rocha-Lima in 1912 (cited by De Monbreun[6]) studied Darling's three cases and concluded that *H. capsulatum* was a fungus closely related to *Cryptococcus farcinosus* which causes epizootic lymphangitis in horses.

Histoplasmosis was considered to be a rare tropical disease until 1926 when Riley and Watson[7] reported a case in a resident of Minnesota. Dodd and Tompkins[8] were the first to diagnose the disease before death by finding the organisms in monocytes of the peripheral blood smears of an infant in Tennessee. From this baby De Monbreun[6] was the first to culture *H. capsulatum* on artificial media. He found that the fungus was dimorphic and could be grown in the mycelial phase on Sabouraud's glucose agar and then be converted to the yeast phase by intravenous inoculation into monkeys. He suggested that the name of the disease should be changed to "cytomycosis of Darling" but this term has not been generally adopted. De Monbreun believed that a saprophytic form of the fungus probably existed free in nature and that insects might prove to be vectors. Later, De Monbreun[9] demonstrated that the dog was a natural host for the fungus and he was able to transmit the disease to dogs by either feeding or inoculation of the organisms. In these animals there were typical chronic granulomatous lesions and the reticulo-endothelial cells contained the yeast phase of the fungus.

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†Present address: Department of Pathology, St. Joseph's Hospital, Hamilton, Ontario.

In 1941 Conant[10] carried out a cultural study of the life cycle of *H. capsulatum* and was able to grow the mycelial phase on Sabouraud's glucose agar at room temperature and to convert it to the yeast phase on sealed blood agar at 37° C. He described the dense mat of branching septate hyphæ which formed a thick, cottony white mycelium on Sabouraud's medium. The aerial hyphæ bore large, thick-walled tuberculate chlamydospores which were the distinguishing morphological characteristic of *Histoplasma capsulatum*. On sealed blood agar, small, convex, light brown, moist colonies grew in 7 to 10 days. In the fresh state the yeast form of the organisms appeared as thin-walled oval bodies, 2 to 3 microns in diameter, which reproduced by a single bud. Each yeast cell contained a large protoplasmic granule and a large vacuole. There was no evidence of encapsulation.

EPIDEMIOLOGY

In 1943 Smith[11] observed that the highest incidence of non-tuberculous pulmonary calcification occurred in those regions of the United States where histoplasmosis was endemic. He suggested that pulmonary histoplasmosis might heal by calcification. In 1945 Christie and Peterson[2] and Palmer,[3] utilizing the histoplasmin skin test,[12, 13] demonstrated that there was a significant relationship between pulmonary calcification and a positive histoplasmin skin test in tuberculin-negative individuals.

An important step in the further understanding of this disease was made in 1949 by Emmons,[14] who was able to culture *H. capsulatum* from the soil where infected rats had been found. He also isolated the organisms from the dog, mouse, rat, cat, skunk, and opossum.[15] Furcolow and Grayston[16] undertook a retrospective survey of 13 minor epidemics of pulmonary disease which had occurred in the United States from 1938 to 1952. These had been called by such terms as "acute miliary pneumonitis", "cave sickness", "unusual type of pulmonary disease" and so on. Serological evidence of histoplasmosis and positive histoplasmin skin tests were found in the individuals involved in these epidemics and, in most cases, *H. capsulatum* was cultured from the soil or locations where the epidemics had occurred. The sites were mostly abandoned buildings or farms, especially where the soil had been contaminated by chicken or pigeon excreta. It would appear that the saprophytic fungus thrives best in soil rich in organic matter, such as in old henhouses and on farms. No reports of the natural occurrence of the organisms in birds have been published and it seems most likely that certain soils are infected from animal carriers. From the infected soil airborne spores are inhaled and the most common route of human infection is via the respiratory tract. This feature makes the laboratory handling of mycelial cultures of *H. capsulatum* quite hazardous and more than 50 cases of infections in laboratory personnel have been reported.[4] Occasional cases occur in humans as a result of ingestion of the spores, or by inoculation into the skin or mucous membranes; but there is no evidence of direct transmission from one person to another.[1]

Although sporadic cases have been reported from many countries of the world, the highest incidence appears to be in the east-central part of the United States,[17, 18] and relatively few cases have been reported from Canada.

CASE REPORTS

The following four fatal cases recently appeared in Southwestern Ontario.

CASE 1.—D.A., an 18-year-old student nurse who had previously lived on a farm in Blenheim, Ontario, was admitted to the Chatham Public General Hospital on December 10, 1953, with chills and fever, pain in the right shoulder and signs of consolidation in the right lung. Six to eight weeks before admission she had coughed up a small amount of blood. Physical examination revealed a pale, exhausted girl with a temperature of 105° F., pulse 120 and respirations 40 per minute. There were no enlarged lymph nodes but the spleen was palpable. A chest film showed calcific densities in the right apex and the hilus of each lung with a region of pneumonitis at the right base. The white cell count was 16,100 (89% neutrophils), red cell count 3,470,000 and platelets 162,000. Serum agglutinins and a blood culture were subsequently reported negative. There was no clinical improvement with Dicrysticin (penicillin and streptomycin) and aureomycin and three days after admission she had a sudden fatal hæmoptysis.

The postmortem examination revealed foci of fibrosis and calcification in the right apex and numerous calcified subpleural nodules in all lobes of the lungs. Patchy subpleural hæmorrhages were present in both lower lobes. The mediastinum contained enlarged caseous and calcified lymph nodes. A definite fistulous tract extended through the matted nodes from the superior vena cava to the right main bronchus. The tract was lined by necrotic grey tissue and contained recent blood clots. The liver was congested and presented numerous miliary calcified nodules beneath the capsule. The spleen was dark red and swollen (350 g.) and the cut sections revealed numerous small calcified nodules. Microscopically, there was a diffuse interstitial fibrosis of the lungs with infiltration by lymphocytes, plasma cells and large mononuclear phagocytes. Many tubercle-like granulomata were present which contained multinucleated giant cells. The tracheobronchial lymph nodes were composed of dense fibrous tissue containing numerous areas of chronic granulomatous inflammation, caseation and calcification. In one region there was a focus of ectopic bone formation. Many of the large mononuclear cells contained *Histoplasma capsulatum*. The mucosa and wall of the right main bronchus were ulcerated at the sites of erosion and the large mononuclear cells of the inflammatory exudate contained typical yeast cells.

H. capsulatum were also present in the reticulo-endothelial cells of the liver and spleen.

CASE 2.—R.D., a 59-year-old male railway employee living in St. Thomas, Ontario, was first admitted to the St. Thomas-Elgin General Hospital in October 1954, because of abdominal pain and diarrhœa. He appeared prematurely old, was easily fatigued and had not worked for some nine years. On admission his hæmoglobin was 6.4 g. %, red cell count 2,650,000 and white cell count 7400. Occult blood was present in the stool on numerous occasions but no pathogenic bacteria were cultured. Serum agglutinins and blood cultures were negative. A laparotomy on October 4 revealed "mesenteric adenitis with generalized hyperæmia, œdema and hypertrophy of the jejunum; fibrinous exudate over surface of small bowel; findings otherwise negative". A lymph node was removed from the mesentery and the pathological report was "granulomatous reaction compatible with regional ileitis". His condition improved considerably with blood transfusions and iron therapy.

He continued to have recurrent diarrhœa and weight loss at home and was readmitted to hospital on June 30, 1955. At this time his hæmoglobin was 3.5 g. %, red cell count 1,960,000 and white cell count 3000. His temperature was 102° F. and pulse rate 120 per minute. He was given whole blood transfusions and streptomycin with no clinical improvement. He developed severe respiratory distress and died three days after admission.

The postmortem examination was performed on an elderly, emaciated man with numerous petechial hæmorrhages and purpuric patches over the back. The jejunum was dilated and congested with œdema of the entire wall and hyperæmia of the mucosa. In the mesentery there were numerous enlarged lymph nodes which contained irregular, firm grey foci and measured up to 3 cm. in diameter. Similar enlarged lymph nodes were present in the mediastinum and neck. The spleen was slightly enlarged (200 g.) and contained scattered, small, firm, grey nodules averaging several mm. in diameter. There was severe serous atrophy of the epicardial fat and brown atrophy of the myocardium indicative of a long-standing chronic debilitating illness. The lungs were slightly œdematous and a few patchy subpleural hæmorrhages were present in all lobes as well as occasional irregular, firm, grey, nodular foci. Microscopically, there was chronic granulomatous inflammation with fibrosis throughout the entire wall of the jejunum and the mesentery. The inflammatory exudate was composed of lymphocytes, plasma cells and large mononuclear cells with occasional giant cell formation. The *H. capsulatum* were not well stained in the routine H. and E. sections but were clearly demonstrated with the periodic acid-Schiff method. Similar lesions were present throughout the lymph nodes, the spleen, the lungs and adventitia of the aorta. No organisms were found in any other viscera or in the bone marrow of the vertebral bodies.

CASE 3.—A.H., a 77-year-old Austrian woman who had been living on a farm near Chatham, Ontario, for several years, was first admitted to the Chatham Public General Hospital on October 1, 1955, with a severe refractory anæmia. While in hospital she developed an acute thyroiditis and was treated with cortisone. At this time the white cell count was 3850 (92% lymphocytes) and sedimentation rate 50 mm. in one hour. She was readmitted on November 9, complaining of several episodes of fever at home. Her hæmoglobin level was 11.9 g. %, red cell count 2,290,000 and white cell count 3500. Her condition improved with blood transfusions. On December 7 she was again admitted to hospital because of an acute tracheobronchitis which responded well to Achromycin (tetracycline). Her final admission was on December 28, 1955, with complaints of recurring fever at home. She was slightly dehydrated and weak, with mottling of the skin over the chest. A chest film showed accentuated lung markings with streaking at both lung bases and congestion apparently of an inflammatory nature. While in hospital her temperature spiked to 104° F. in the afternoons. A blood culture was eventually reported as negative. Her condition gradually deteriorated and she died on January 5, 1956.

Permission for a postmortem examination was limited to the sternal bone marrow. Microscopically, the marrow was fatty with marked reticulo-endothelial hyperplasia and contained tiny granulomata resembling tubercles. Epithelioid cells and Langhans' type of giant cells were present within these lesions. Practically all of the reticulo-endothelial cells were filled with *H. capsulatum*. There was a notable decrease in both the myeloid and erythroid elements. Although no other organs could be examined, this was almost certainly a case of acute disseminated histoplasmosis.

CASE 4.—H.M., a 59-year-old policeman living in St. Thomas, Ontario, was admitted to the St. Thomas-Elgin General Hospital on July 5, 1955, with difficulty in breathing, shortness of breath, loss of appetite and weakness. His pulmonary symptoms began in July 1954 and gradually became more severe. By December he had developed a chronic cough and was producing 2 to 3 oz. of sputum daily. Physical examination on admission revealed diminished air entry into the right upper lobe with rales and rhonchi present throughout the entire chest. A radiograph showed infiltration of the right apex suggestive of pulmonary tuberculosis. Three sputum cultures were negative for tubercle bacilli. He was sent to the Beck Memorial Sanatorium for further investigation and treatment. A chest film on July 27, 1955, showed increased density and cavitation of the right upper lobe. In November a histoplasmin skin test was positive (1:1000) and *Histoplasma capsulatum* was cultured from the sputum (Figs. 1 to 4). Several subsequent sputum cultures were also positive. By February 1956, there was widespread infiltration of the left

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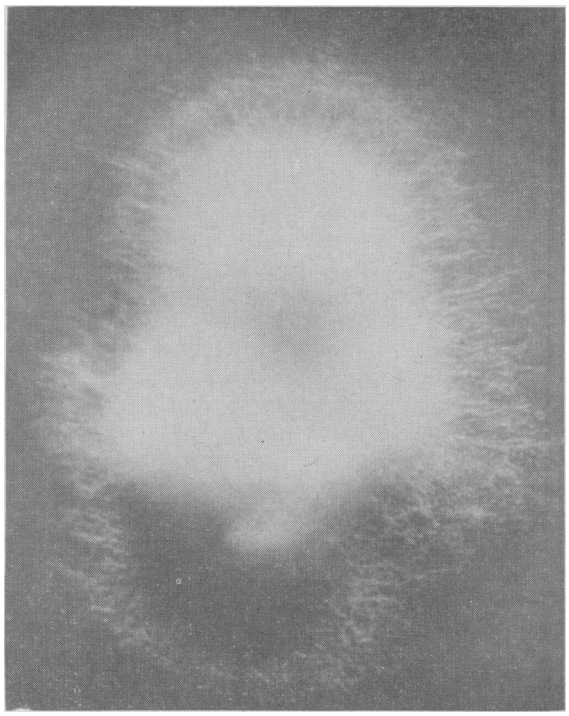

Fig. 1.—Large cottony white colony of the mycelial phase of *H. capsulatum* grown on Sabouraud's glucose agar at room temperature. × 4.

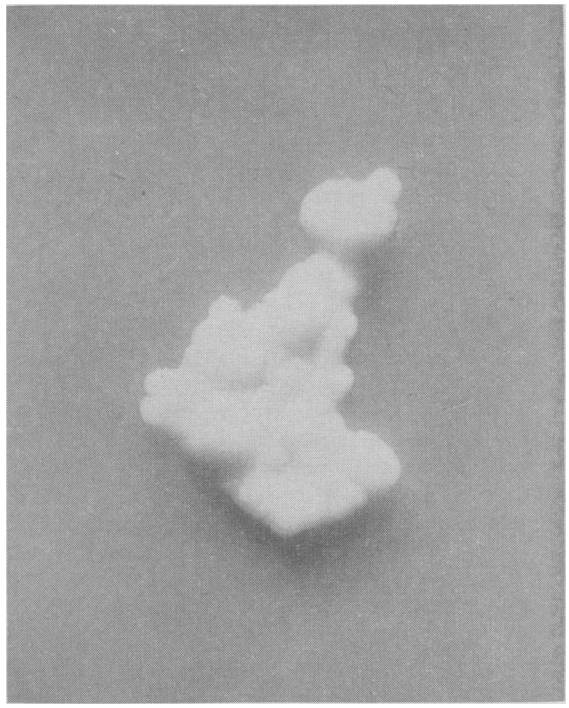

Fig. 2.—Large moist pale yellowish-white colony of the yeast phase of *H. capsulatum* grown on sealed blood agar at 37° C. × 4.

lung and his condition steadily deteriorated. He died on March 21, 1956.

A postmortem examination was performed by Dr. R. C. Buck. Dense fibrous adhesions were present in each pleural cavity and both lungs were enlarged and heavy. In each apex there were large irregular cavities which contained semi-fluid, white purulent material and were surrounded by dense fibrous tissue. No significant lesions were present in the other organs. Microscopically, there were

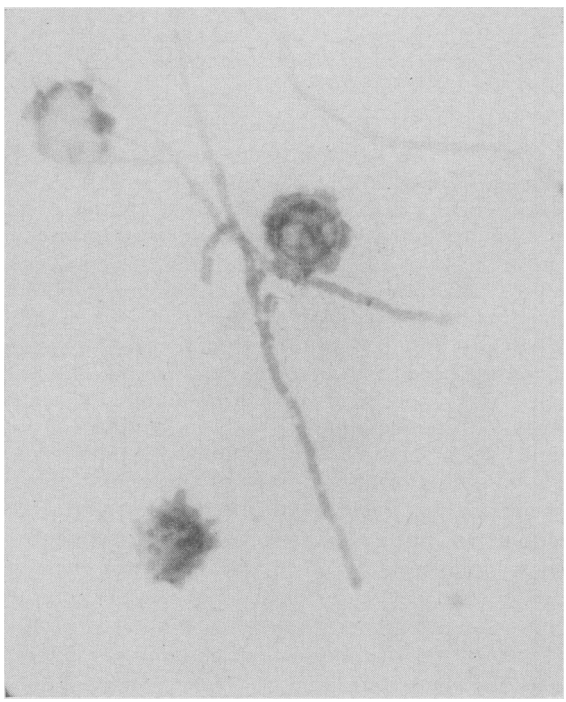

Fig. 3.—Characteristic septate hyphæ and tuberculate chlamydospores from a culture of the mycelial phase of *H. capsulatum*. Gram's stain. × 1500.

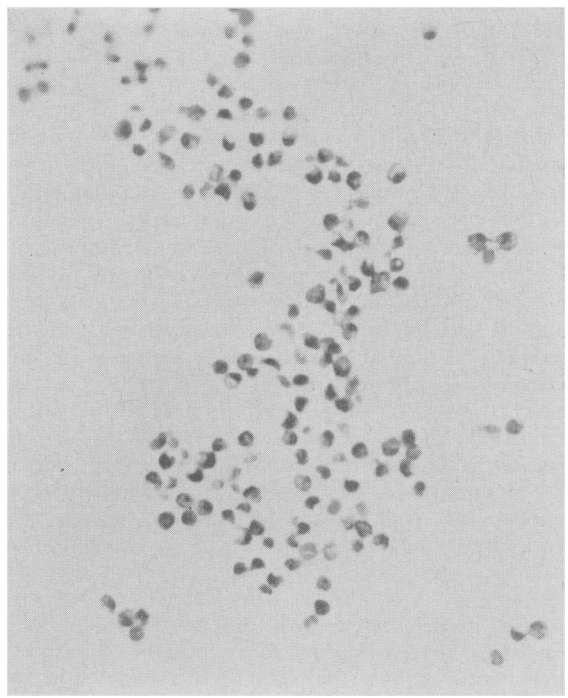

Fig. 4.—Characteristic yeast phase micro-organisms from a culture of *H. capsulatum* illustrating the eccentric chromatin and clear cytoplasm. Air dried, Wright's blood stain. × 960.

extensive foci of necrosis and fibrosis throughout both upper lobes and the right middle lobe. At the margins of these regions, and also present within the fibrous tissue, there were foci of chronic granulomatous inflammation containing numerous multinucleated giant cells. Although no *H. capsulatum* were visible in the routine H. and E. sections, the organisms were revealed in the large phagocytic cells by the periodic-acid Schiff stain. In the adjacent lung tissue there was extensive interstitial fibrosis and many of the alveoli were filled with chronic inflammatory cells. No other organs were found to be involved.

In addition to these four cases, three fatal cases of acute disseminated histoplasmosis have occurred in London, Ontario.[19] This is a total of seven fatal cases of histoplasmosis in Southwestern Ontario during the past few years. There is also a fairly large group of patients in this district in which a diagnosis of benign pulmonary histoplasmosis has been made during recent months.

There has been an increasing number of published reports of histoplasmosis in Canada during the past few years. In 1950, Green[20, 21] mentioned a fatal case in Winnipeg, Manitoba, and also described a case of benign pulmonary histoplasmosis in a 24-year-old man who had visited Southern Ontario before his illness. The first detailed account of acute disseminated histoplasmosis in Canada was that of Blanchard[22] in 1951. The patient was a 69-year-old man from Toronto, Ontario. In 1953 Jones[23, 24] described a fatal case in a 69-year-old man from Kingston, Ontario. He also reported several examples of the benign pulmonary form of histoplasmosis, indicating that the disease was not uncommon in Southeastern Ontario. Mankiewicz, Blank and Rubin[25] in 1954 reported a non-fatal case of pulmonary histoplasmosis in a 31-year-old man from Montreal, Quebec. The diagnosis was proven by culture of the causative organs from the sputum. Nuttall-Smith[26] reported a case of benign pulmonary histoplasmosis in a 13-year-old girl from Kamloops, B.C., in 1956. There was no actual proof of the disease and the diagnosis has been questioned.[27] In 1956, Fish, Schroder and Fischer[28] described a case of histoplasmosis in a dog in Guelph, Ontario. In the same year, Karnauchow and Marciniak[29] reported a fatal case in a 13-year-old girl in Ottawa, Ontario, and Grant[30] reported a case of benign pulmonary histoplasmosis in a 34-year-old man from Toronto, Ontario. He stated that in various Toronto hospitals there were four recorded cases of active disease and several examples of asymptomatic histoplasmosis. Fischer *et al.*[31] stated, "Serological evidence suggests that primary histoplasmosis is not uncommon in Southern Ontario and that it should be considered in the differential diagnosis of pulmonary infections." Unfortunately, no details are given as to actual incidence.

TABLE I.—HISTOPLASMIN SKIN SENSITIVITY IN CANADA

Location	*Year*	*Reference*	*Type of individuals*	*Numbers tested*	*% reactors*
Beloeil, Quebec	1949	32	School children	75	26.7
Halifax, Nova Scotia	1949	33	University students	310	1.3
Newfoundland	1949	33	General population	157	0
Abitibi-East, Northern Quebec	1949	34	Indians	161	5
St. Agathe des Monts, Quebec	1950	17	Sanatorium patients	270	10.4
Winnipeg, Manitoba	1950	20, 21	Service Veterans Hospital	440	5.2
Toronto, Ontario	1950	35	University students with pulmonary calcification	63	57.2
Kingston, Ontario	1953	24	University students	?	20 to 30
Toronto, Ontario	1955	36	University students with no pulmonary calcification	134	9.7
Montreal, Quebec	1955	37	Patients referred to Chest Clinic	100	21

In addition to the above case reports in Canada, several surveys have been made using the histoplasmin skin test.[17, 20, 21, 24, 32-37] These results are summarized in Table I.

From Table I it can be seen that the highest incidence of reactors in a random population sample is in Southern Quebec, near the St. Lawrence River. A moderate number of reactors was found in Kingston, which is on the north shore of Lake Ontario near the source of the St. Lawrence River. There was a high number of reactors among Toronto university students in 1950 because only those cases with pulmonary calcification were tested. However, this group included residents from every Canadian province except Newfoundland and Prince Edward Island. This indicates that the disease may occur in widely separated regions of Canada and that more histoplasmin surveys should be made across the nation.

One of us (E.L.B.) has now skin-tested 400 people selected at random from St. Thomas and

the surrounding district. These results are listed in Table II. All tests were made with standardized Histoplasmin (Parke, Davis Co.) 1:100 and read in 48 hours. A circular zone of erythema and induration measuring 1 cm. or more in diameter was considered a positive test. The incidence is unusually high and compares with some of the regions of highest endemicity of histoplasmosis in the United States.[4] In view of the fact that seven fatal cases of this disease have been recognized in Southwestern Ontario, the high incidence of positive skin reactors is not surprising. From various studies in the United States it would appear that there are many thousands of cases of the benign healed pulmonary form of histoplasmosis for every one that terminates fatally because of dissemination or complications.

TABLE II.—HISTOPLASMIN SKIN SENSITIVITY IN ST. THOMAS, ONTARIO

Sex	*Year*	*Type of individuals*	*Number tested*	*% reactors*
Male	1956–57	Random population	172	79.7
Female	1956–57	Random population	228	78.9
			Total: 400	79.3

Histoplasmosis appears to be a relatively new disease in this part of Canada, and this may not merely mean that the disease is now being looked for and recognized. One possible factor in this regard may be the increasing use of antibiotics, since it has been shown that the *in vitro* growth of *Histoplasma capsulatum* is enhanced by some of the antibiotics, among them streptomycin.[38] If a clinical case of unrecognized benign pulmonary histoplasmosis is treated with streptomycin in the mistaken belief that it is tuberculosis, then it is theoretically possible that the growth of *H. capsulatum* could be enhanced and result in acute dissemination of the disease and death. It might be thought that cortisone, too, may result in the breaking down of healing pulmonary lesions of benign histoplasmosis and cause dissemination, but this has not been supported by experimental infections in mice.[39]

TABLE III.—AGE DISTRIBUTION OF HISTOPLASMIN REACTORS

Age	*Males*			*Females*		
	Negative	*Positive*	*% reactors*	*Negative*	*Positive*	*% reactors*
1 - 9	13	25	65.7	11	6	35.3
10 - 19	3	8	72.7	6	19	76.0
20 - 29	5	34	87.1	11	70	86.4
30 - 49	9	58	86.5	12	66	87.2
50 plus	5	12	70.6	8	19	70.4

The age distribution of the reactors to histoplasmin is listed in Table III. There were only about 30 pre-school children in the series and these were not listed as a separate group. However, of these there were only about 30% with a positive reaction. The lowest incidence is in childhood, with a sharp rise to a maximum in young adults. In the lower age group there are more reactors among the boys than the girls, which may reflect the preference of the former to play in dirt. Among the teen-age group and adults there is no apparent sex difference in positive skin reactors. Other incomplete studies of histoplasmin reactors in regions within 20 miles of St. Thomas have not yielded as high a number of positives. This has also been the finding in several regions of the United States where a high incidence of positive reactors has been found in one end of a county and a low incidence in the other.

It is our belief that a greater effort should be made to diagnose this disease in clinical practice and more use made of sputum cultures, the complement fixation test and the histoplasmin skin test. More histoplasmin skin test surveys are required from all parts of the country in order to map out the epidemiological picture of histoplasmosis in Canada.

SUMMARY

The historical background of histoplasmosis is reviewed and a total of seven cases from Southwestern Ontario are reported. All the known reported cases in Canada are mentioned, together with a tabulation of all the known histoplasmin skin sensitivity surveys made in this country. To this group are added the results of 400 tests done on a random population in the St. Thomas region, where an incidence of 79.3% positive skin reactors has been

found. Histoplasmosis should be considered in the differential diagnosis of any obscure pulmonary disease, and suspect cases should probably not be treated with antibiotics or cortisone because of the possibility of causing dissemination and death. More histoplasmin surveys are needed from other parts of Canada.

We wish to acknowledge the assistance of Mr. A. W. Hambleton, who grew the cultures used in the illustrations, and of Mr. C. E. Jarvis, who took the photomicrographs. Dr. J. C. Paterson kindly consented to the use of Case 4. We should like to thank all the pathologists in Southwestern Ontario for their kindness in answering queries regarding histoplasmosis in their districts.

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RÉSUMÉ

Après avoir rappelé les données historiques de l'histoplasmose, les auteurs soulignent le fait que des cas sporadiques ont été rapportés ici et là de la plupart des provinces canadiennes, et que la plus grande agglomération d'individus à cuti-réaction positive se trouve dans la région de Beloeil le long du fleuve. La fréquence des réactions positives, faible durant l'enfance, subit une élévation soudaine pendant l'adolescence pour atteindre un sommet chez les jeunes adultes. La nouveauté de cette affection au Canada ne vient pas seulement des efforts accrus déployés pour son dépistage, mais pourrait bien découler de l'usage grandissant des antibiotiques, car il ressort de certains travaux que la croissance de *H. capsulatum* est favorisée *in vitro* par la présence de streptomycine. Les faits cliniques de quatre cas fatals de cette affection sont présentés dans le texte. Ces malades venaient du sud-ouest de l'Ontario. Les résultats d'une enquête menée par la suite dans la région de Saint-Thomas (Ontario) ont révélé une endémicité insoupçonnée.

VALUE OF BRONCHOGRAPHY IN THE DIAGNOSIS OF PULMONARY DISEASES*

J. HALLE, M.D., L. MONTMINY, M.D., G. PARADIS, M.D. and M. BILODEAU, M.D.,† *Quebec, Que.*

PULMONARY PATHOLOGY is intricate and often filled with surprises. Diagnosis and treatment necessitate a complete investigation, both anatomical and physiological, of the respiratory apparatus. Care and precision in studying the nature and location of pulmonary disease are mainly responsible for the effectiveness of its treatment. Radiological, bacteriological and clinical evaluation are essential, but one cannot do without the aid of bronchoscopy and bronchography. Bronchoscopy permits the visualization of the main lobar and segmentary openings, and checks the appearance of the mucosa; it can also detect an abnormal endobronchial process or a lesion of the related lobes and segments. However, only bronchography will confirm or delineate such a lesion (Fig. 1).

Recent years have witnessed a great improvement in the technique of bronchography. The contrast medium can now be directed through a catheter towards any definite area, hence the procedure is easier and safer. The water-soluble contrast agents are rapidly eliminated from the bronchial tree and interfere neither with the radiological study nor with the surgical schedule.

Bronchography gives accurate information on the size and relative location of the bronchi and can reveal congenital or other anomalies. It is

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†Laval Hospital, Quebec, Que.