Treatment of Bronchogenic Carcinoma with Nitrogen Mustard*

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There have been numerous reports as to the clinical results obtained with the chloroethylamines (nitrogen mustards) in the treatment of Hodgkin's disease, lymphosarcoma and leukemia.1-11,19 There has been a paucity of reported clinical trials in regard to the treatment of bronchogenic carcinoma with nitrogen mustard despite the fact that a few cases of carcinoma of the lung treated with nitrogen mustard have responded to therapy. Rhoads12 states that cancer of the lung can be caused to regress temporarily in about 50 per cent of the instances and that these effects are transient and incomplete. The largest series of cases has been reported by Boyland, et. al.13 Their series included 41 histologically proved cases of bronchogenic carcinoma which were given methylbis-(B chloroethyl) amine. These cases were unsuitable for other forms of therapy. Symptomatic relief and objective signs of improvement were noted in approximately one-half of them. Karnofsky has reported temporary symptomatic remissions in four patients with anaplastic carcinoma of the lung.14 Skinner, et. al.15 reported treating 25 cases of bronchogenic carcinoma with nitrogen mustard and was of the opinion that clinical improvement was noted in about 70 per cent for periods ranging from six weeks up to six months, after which time the drug became ineffective. Ben-Asher9 published a report on 11 patients with carcinoma of the lung treated with nitrogen mustard, nine of whom did not respond to therapy. Wintrobe and Huguley10 treated four cases of bronchogenic carcinoma; two of them had a fair response to therapy. Gellhorn and Jones state in their review, Chemotherapy of Malignant Diseases,11 that some favorable therapeutic responses have been observed using nitrogen mustard in the treatment of anaplastic carcinoma of the lung, the exact significance of which must remain unsettled until further clinical trials can be undertaken.

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Materials and Methods

In our study we used methyl-bis-(B-chloroethyl) amine. The history, chemistry and pharmacodynamics of the drug have been discussed in detail by others.\textsuperscript{16-18} The clinical evaluation of each patient prior to therapy included a careful history, physical examination, evaluation of previous therapy, and confirmation biopsies.

Roentgenographic studies of the chest were made on all patients, and studies of skeletal areas were made when indicated. Studies of the peripheral blood routinely included: hemoglobin determination, red blood cell count, white blood cell count, platelet count and a differential leucocyte count. Other laboratory examinations were done when they appeared indicated.

The nitrogen mustard was given intravenously through the rubber tubing of an intravenous drip of normal saline solution. A course consisted of four injections of the drug. A daily dose of 0.10 mg. per kilogram of body weight was administered.

A hematological study, such as was obtained prior to therapy, was performed every second day during treatment and usually twice a week thereafter. The patient was seen daily and examined at least weekly. Roentgen studies were made as deemed necessary.

Results

A total of 19 male patients were treated with nitrogen mustard. The pertinent data of each patient treated are tabulated in Table 1. We have attempted to evaluate the effect of HN2 (nitrogen mustard) as good, fair or poor. The response has been classified as good when the patient was kept comfortable and free of incapacitating symptoms for six months or more. When the patient had comparable improvement for a shorter period or had fair symptomatic improvement for a longer period (approximately six months), or had marked relief of a serious symptom even though the course of the disease was not altered, the response has been termed fair. When improvement was short-lived or slight, the response has been classified as poor.

The average age of the patients in this series of cases was 54.42 years, the range being from 29 to 68 years. The duration of symptoms prior to HN2 averaged 7.47 months, the shortest time interval being one month and the longest 13 months. Seven of the 19 patients had an exploratory thoracotomy prior to HN2 therapy and were inoperable, three of the 19 had x-ray therapy with poor results. One patient had both x-ray and thoracotomy before treatment was initiated. Ten of the 19 patients had no treatment before HN2. A summary of the results of treatment is given in Table II. Five of the seven patients still living have anaplastic carcinoma and two have squamous cell carcinoma.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Sex and Age</th>
<th>Duration Prior to HN2</th>
<th>Previous Therapy</th>
<th>Condition Prior to HN2</th>
<th>No. of Courses</th>
<th>Total Dose Mg.</th>
<th>Effect</th>
<th>Other Therapy</th>
<th>Time Since 1st HN2</th>
<th>Present Condition and Comment</th>
<th>Final Evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M*, 62</td>
<td>8</td>
<td>Surgery</td>
<td>Inop.</td>
<td>Fair</td>
<td>17.2</td>
<td>Fair</td>
<td>None</td>
<td>8 mos.</td>
<td>Living</td>
<td>Fair</td>
</tr>
<tr>
<td>2</td>
<td>M, 54</td>
<td>13</td>
<td>Surgery</td>
<td>Inop.</td>
<td>Very poor</td>
<td>25.2</td>
<td>Poor</td>
<td>None</td>
<td>1 wk.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>3</td>
<td>M, 54</td>
<td>12</td>
<td>Surgery</td>
<td>Inop.</td>
<td>Poor</td>
<td>3½ 97.4</td>
<td>Good</td>
<td>X-ray</td>
<td>9 mos.</td>
<td>Living</td>
<td>Good</td>
</tr>
<tr>
<td>4</td>
<td>M*, 55</td>
<td>10</td>
<td>None</td>
<td>...</td>
<td>Very poor</td>
<td>24.0</td>
<td>Poor</td>
<td>None</td>
<td>1 mo.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>5</td>
<td>M, 49</td>
<td>10</td>
<td>None</td>
<td>...</td>
<td>Fair</td>
<td>25.6</td>
<td>Poor</td>
<td>None</td>
<td>5 mos.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>6</td>
<td>M, 56</td>
<td>4</td>
<td>X-ray</td>
<td>Poor</td>
<td>Poor</td>
<td>21.8</td>
<td>Poor</td>
<td>None</td>
<td>1 mo.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>7</td>
<td>M, 56</td>
<td>6</td>
<td>None</td>
<td>...</td>
<td>Poor</td>
<td>26.0</td>
<td>Poor</td>
<td>None</td>
<td>3 wks.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>8</td>
<td>M, 57</td>
<td>9</td>
<td>Surgery</td>
<td>Inop.</td>
<td>Poor</td>
<td>29.6</td>
<td>Poor</td>
<td>None</td>
<td>1 mo.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>9</td>
<td>M, 52</td>
<td>11</td>
<td>Surgery</td>
<td>Inop.</td>
<td>Poor</td>
<td>25.6</td>
<td>Poor</td>
<td>None</td>
<td>5 wks.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>Case No.</td>
<td>Sex and Age</td>
<td>Duration Prior to HN2</td>
<td>Previous Therapy</td>
<td>Condition Prior to HN2</td>
<td>No. of Courses</td>
<td>Total Dose Mg</td>
<td>Effect</td>
<td>Other Therapy</td>
<td>Time Since 1st HN2</td>
<td>Present Condition and Comment</td>
<td>Final Evaluation</td>
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<tr>
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<td>---------------------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>10</td>
<td>M 67</td>
<td>8</td>
<td>None</td>
<td>Poor</td>
<td>1</td>
<td>23.2</td>
<td>Fair</td>
<td>None</td>
<td>3 mos.</td>
<td>Living</td>
<td>Fair</td>
</tr>
<tr>
<td>11</td>
<td>M 60</td>
<td>7</td>
<td>None</td>
<td>Very poor</td>
<td>2</td>
<td>46.4</td>
<td>Good</td>
<td>None</td>
<td>6 mos.</td>
<td>Living</td>
<td>Good</td>
</tr>
<tr>
<td>12</td>
<td>M 33</td>
<td>8</td>
<td>X-ray</td>
<td>None</td>
<td>2</td>
<td>58</td>
<td>Poor</td>
<td>None</td>
<td>2 mos.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>13</td>
<td>M 60</td>
<td>4</td>
<td>None</td>
<td>Very poor</td>
<td>1</td>
<td>21.7</td>
<td>Poor</td>
<td>None</td>
<td>1 mo.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>14</td>
<td>M 68</td>
<td>6?</td>
<td>None</td>
<td>Fair</td>
<td>1</td>
<td>18.0</td>
<td>Poor</td>
<td>None</td>
<td>1 mo.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>15</td>
<td>M 62</td>
<td>10</td>
<td>Surgery X-ray</td>
<td>Inop.</td>
<td>1</td>
<td>20.8</td>
<td>Poor</td>
<td>None</td>
<td>2 mos.</td>
<td>Living</td>
<td>Poor</td>
</tr>
<tr>
<td>16</td>
<td>M 53</td>
<td>1</td>
<td>None</td>
<td>Very poor</td>
<td>1</td>
<td>24</td>
<td>Poor</td>
<td>None</td>
<td>4 days</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>17</td>
<td>M 60</td>
<td>5</td>
<td>Surgery</td>
<td>Inop.</td>
<td>1</td>
<td>24.8</td>
<td>Poor</td>
<td>None</td>
<td>1 mo.</td>
<td>Dead</td>
<td>Poor</td>
</tr>
<tr>
<td>18</td>
<td>M 29</td>
<td>2</td>
<td>None</td>
<td>Fair</td>
<td>1</td>
<td>32</td>
<td>Poor</td>
<td>None</td>
<td></td>
<td>Living</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>M 57</td>
<td>6</td>
<td>None</td>
<td>Good</td>
<td>1</td>
<td>32</td>
<td>None</td>
<td>None</td>
<td></td>
<td>Living</td>
<td></td>
</tr>
</tbody>
</table>

*Negro.
The toxic effects of nitrogen mustard when used as a chemotherapeutic agent has been frequently and adequately emphasized by others.\textsuperscript{3-7,11,17-19} In this series of cases similar toxic effects were noted. Nausea and vomiting occurred in a high percentage of the cases within one to eight hours following therapy. Nitrogen mustard therapy often resulted in a decrease in the leucocyte count and platelet count, but in no patient did serious complications arise as the result of these effects.

\textbf{CASE REPORTS}

\textbf{Case 3}: K.F., male, age 53 years. \textit{History}: This patient was admitted to the hospital for the first time on July 29, 1948 with the history of frequent attacks of dyspnea for one year prior to entry. Seven weeks prior to admission he had slight hemoptysis, followed in a few days by hoarseness and severe pain in the right anterior chest at the level of the second interspace and radiating to the sternum. Three weeks prior to entry he

\begin{table}
\centering
\caption{Summary of Results of Treatment with Nitrogen Mustard}
\begin{tabular}{lcc}
\hline
Total Number of cases & & 19 \\
Male & & 19 \\
Female & & 0 \\
\hline
Average age (years) & & 54.42 \\
Range & & (29 to 68) \\
\hline
Average duration of disease (months) & & 7.47 \\
Range (Prior to HN2) & & (1 to 13 mos.) \\
\hline
Number explored surgically & & 7 \\
Number previously treated with x-ray & & 3 \\
Number initially treated with HN2 & & 10 \\
Number of courses given (Range) & & (1/2 to 3 1/2) — (total 23 1/2) \\
\hline
Condition prior to HN2: & \begin{tabular}{l|c}
1) Good & 1 \\
2) Fair & 5 \\
3) Poor & 8 \\
4) Very poor & 5 \\
\end{tabular} & \begin{tabular}{l|c|c}
No. Living & Effect of HN2* \\
1 & 1 & 2 \\
2 & 2 \\
3 & 13 \\
4 & 1 \\
\end{tabular} \\
\hline
*Two patients treated too recently to be evaluated. \\
\hline
Number patients living since HN2 started & & 7 \\
Months living since HN2 started & & 2-9 \\
\hline
Number patients dead since HN2 started & & 12 \\
a) Length of life following HN2 & & 2 days to 5 months \\
\hline
Final evaluations:* & \begin{tabular}{l}
1) Good \\
2) Fair \\
3) Poor \\
\end{tabular} & \begin{tabular}{l}
2 \\
2 \\
13 \\
\end{tabular} \\
\hline
*Two patients treated too recently to be evaluated. \\
\end{tabular}
\end{table}

One patient received x-ray therapy following HN2.
had an episode of coughing and developed severe pain in the left antecubital fossa which radiated to his left shoulder; within a half hour his left arm began swelling and within two hours it was greatly enlarged. He lost 20 lbs. in two months. Past history revealed that he was discovered to have positive serologic test for syphilis 20 years prior to entry and had received antisyphilitic therapy intermittently for two years.

*Physical Examination:* Weight 159 lbs., B.P. 120/80 R.A., 130/100 L.A. He appeared chronically ill. His left pupil was larger than the right.

### TABLE 3: Patients Still Alive

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Type of Carcinoma</th>
<th>Comment / Clinical Improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Anaplastic (suggestive of epidermoid)</td>
<td>Slight gain in weight.</td>
</tr>
<tr>
<td>3</td>
<td>Anaplastic</td>
<td>Edema of face, arms and legs—disappeared with relief of pain following 1st course (relief of caval obstruction). Following 2nd course similar effect.</td>
</tr>
<tr>
<td>10</td>
<td>Squamous</td>
<td>Gained 6 lbs. weight, improved appetite.</td>
</tr>
<tr>
<td>11</td>
<td>Undifferentiated type</td>
<td>Gained 21 lbs. weight and energy. Increased appetite.</td>
</tr>
<tr>
<td>15</td>
<td>Anaplastic</td>
<td>No improvement. Weight loss.</td>
</tr>
<tr>
<td>18</td>
<td>Poorly differentiated epidermoid</td>
<td>Treated too recently to be evaluated.</td>
</tr>
<tr>
<td>19</td>
<td>Squamous</td>
<td>Treated too recently to be evaluated.</td>
</tr>
</tbody>
</table>

### TABLE 4: Patients Dead

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Type of Carcinoma</th>
<th>Length of Life Following HN2</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Epidermold</td>
<td>1 week</td>
</tr>
<tr>
<td>4</td>
<td>Anaplastic</td>
<td>1 month</td>
</tr>
<tr>
<td>5</td>
<td>Poorly differentiated epidermoid</td>
<td>5 months</td>
</tr>
<tr>
<td>6</td>
<td>Anaplastic</td>
<td>1 month</td>
</tr>
<tr>
<td>7</td>
<td>Anaplastic</td>
<td>3 weeks</td>
</tr>
<tr>
<td>8</td>
<td>Poorly differentiated epidermoid</td>
<td>1 month</td>
</tr>
<tr>
<td>9</td>
<td>Undifferentiated</td>
<td>5 weeks</td>
</tr>
<tr>
<td>12</td>
<td>Poorly differentiated epidermoid</td>
<td>2 months</td>
</tr>
<tr>
<td>13</td>
<td>Undifferentiated</td>
<td>1 month</td>
</tr>
<tr>
<td>14</td>
<td>Anaplastic</td>
<td>1 month</td>
</tr>
<tr>
<td>16</td>
<td>Undifferentiated</td>
<td>4 days</td>
</tr>
<tr>
<td>17</td>
<td>Epidermold</td>
<td>1 month</td>
</tr>
</tbody>
</table>
right jugular vein was prominent. There was dulness to percussion over the left base posteriorly with decreased breath sounds over this same area. Dilated veins were visible over the left antero-thoracic cage and over the left arm. The left arm was edematous from his shoulder to his wrist. His legs and face were also edematous. Venous pressure determinations on August 3, 1948 revealed a V.P. of 375 mm. citrate in the right antecubital vein and 140 mm. citrate in the left. Circulation time with decholin was 26 seconds, with ether 14.5 seconds. Kahn and Wassermann tests were both reported as doubtful.

Roentgenogram and fluoroscopy of chest on July 28, 1948 revealed a soft tissue mass occupying the anterior superior mediastinum and was interpreted as an aneurysm of the ascending arch of the aorta.

Because of the previous history of antisyphilitic therapy, doubtful serologic findings and diagnosis of aortic aneurysm, antiluetic treatment was begun. He received penicillin 5,000 units intramuscularly every three hours for three days and then 50,000 units every three hours for 15 days. He was seen by the consultant in Chest Surgery who recommended exploratory thoracotomy with the consideration of possibly wrapping the aneurysm with polythene cellophane. However on August 21, 1948 he was again fluoroscoped and no pulsation of the mediastinal mass was observed.

Exploratory Thoracotomy was performed on August 23, 1948 and a large inoperable nodular tumor was found in the mediastinum about the great vessels especially beneath the arch of the aorta and carotid. Biopsy was done.

Pathological Report: Anaplastic carcinoma of bronchogenic origin.

Treatment: Nitrogen mustard was administered (7.6 mg. per day) for four days beginning September 4, 1948. By September 11 edema of the legs was greatly improved, and the face and arms were free of edema.

FIGURE 1. Case 3-A, March 9, 1949: Atelectasis right upper lobe with marked widening of the mediastinum.—Case 3-B, April 9, 1949: After HN2 therapy, regression of atelectasis, right upper lobe and diminution of the mediastinal widening. This was associated with marked clinical improvement.
X-ray film of chest on September 17 did not reveal the previously noted soft tissue mass in the anterior mediastinum although there was some increase in density in the area it previously occupied. Pain was relieved by small amounts of codeine and cough and hoarseness following therapy were less. He was discharged from the hospital on October 7, 1948 and was seen several times before further therapy was indicated. He remained comfortable except for occasional epistaxis and dull chest pain which was relieved by codeine and aspirin. Three weeks before readmission or three months after HN2, he again became hoarse, and one week before entry he had severe chest pain, hemoptysis and pedal edema. He was readmitted to the hospital on December 6, 1948 when he weighed 142 lbs. X-ray inspection of chest showed little if any change. He received 6.5 mg. of nitrogen mustard per day for four days beginning December 8th.

The ankle edema again disappeared and there was no further hemoptysis. X-ray film of chest on December 21 revealed no further evidence of a mediastinal mass. He was discharged from the hospital on January 5, 1949 and was seen several times thereafter. During the interval before readmission he was again fairly comfortable; two months before re-entry he began to note dyspnea on exertion (this was one month after second course of nitrogen mustard). However, dyspnea did not become severe until one week before entry. Three days before admission he had ankle edema, hemoptysis, hoarseness and venous distension over chest. He was hospitalized on March 8, 1949 when he weighed 143 1/2 lbs. X-ray inspection of the chest on March 9 revealed a homogenous increase in density in the upper one-half of the right lung field and increase in density in the left suprahilar region. He received 6.5 mg. nitrogen mustard per day for four days beginning March 10.

Dyspnea became greatly improved and he gained two pounds following therapy. His appetite, energy and feeling of well being all increased. X-ray inspection of chest on April 9, 1949 still revealed some widening of the mediastinal shadow but appeared diminished in extent. The previous density in the upper half of the right lung field had completely disappeared. Symptomatic remission was brief however and on April 21 there was considerable swelling of the neck. There was also noted distension of the superficial veins of the neck, chest and abdomen. X-ray film of chest on April 21 revealed widening of the superior mediastinum with possibly slight increase in the width of the mediastinum compared to previous x-ray films. The lungs were still clear.

He was given 6.5 mg. nitrogen mustard per day for two days beginning April 23, 1949. This brief course of therapy was given because of the short interval since his full course of treatment. The effect again was good but transient; because of this and his discouragement, it was felt that a trial of x-ray therapy was indicated. This was instituted on May 11, 1949. The neck vein distension disappeared after four treatments and he was more comfortable. X-ray inspection of chest on June 15, 1949 revealed reduction in the size of the mediastinal mass.

Case 2: S.H., male, age 60 years. History: This man was admitted to the hospital on January 6, 1949 with the history of frequent pains in the chest and shoulders since June, 1948. He developed generalized edema and lost 67 lbs. of weight in six months. In October, 1948, he had a productive cough, occasional hemoptysis and became dyspneic. Shortly before entry he began to regurgitate food through his nose and had difficulty in swallowing.
On examination he weighed 123 lbs, B.P. 140/80. He was poorly nourished, appeared acutely ill, spoke hoarsely, and was weak. There was limitation of motion of both sides of the chest, more marked on the right. There was dulness to percussion over the right base with absent breath sounds over this area. The liver was felt two to three fingers breaths beneath the right costal margin and was slightly tender. There were slight edema of legs and slightly enlarged bilateral inguinal and axillary nodes.

X-ray inspection of chest on January 10, 1949 revealed a homogenous increase in density of the right base. An infiltrative process was visualized radiating outward and downward from the right hilum region. Fluoroscopy of the esophagus and chest showed an irregular defect in the cervical portion of the esophagus which also involved the hypopharynx. Puddling in the pyriform sinus indicated esophageal obstruction. On January 21 esophagoscopy was negative. and bronchoscopy showed the presence of a soft nodular mass in the right middle lobe orifice. Biopsy revealed undifferentiated bronchogenic carcinoma.

It was agreed that the bronchogenic carcinoma had probably spread to the mediastinum and had caused obstruction by extrinsic pressure on the esophagus and therefore was inoperable. Therefore HN2 5.6 mg. per day was administered for four days beginning January 26, but he gradually developed further esophageal obstruction and suction was required to remove accumulation of mucus. Cough increased in severity. Dyspnea increased and he became weak and practically bed-ridden. X-ray film of chest on March 31, 1949 showed a moderate increase in the infiltrative process involving the right lower lung field. Swallowed barium failed to reveal any definite evidence or narrowing of the esophagus. His weight was 123 lbs. He received 6.0 mg. HN2 per day for four days beginning March 28, 1949.

His condition was poor at the beginning of nitrogen mustard therapy, but his appetite improved and he gradually gained weight and energy. The dyspnea improved and he was able to swallow solids. He left the hospital on leave on April 15 and felt so improved that he desired to remain at home. He returned on May 12 when he felt extremely well and weighed 135 lbs. X-ray film of chest on May 15 showed the previously reported area of increased density in the region of the right middle lobe. Swallowed barium did not reveal any disease of the esophagus. He was discharged on May 21. On June 21, 1949 he felt considerably stronger, his appetite had remained good and he had gained 3 lbs. in weight in the month following discharge from the hospital. He had not had any further symptom of esophageal obstruction.

**SUMMARY**

1) The effects of methyl-bis-(B chloroethyl) amine in 19 patients with histologically proved bronchogenic carcinoma are described. These cases were unsuitable for other forms of therapy.

2) Seven of the 19 patients are still alive. Five of the seven living have anaplastic carcinoma.

3) A good effect was obtained in two patients having anaplastic carcinoma. One is still living nine months after three and one-half courses of nitrogen mustard therapy. The other patient is
still living six months following two courses of HN2. Both had superior mediastinal involvement with decrease in tumor size and symptoms following therapy.

4) Nitrogen mustard often resulted in decrease in the leukocyte count, and platelet count, but in no patient did serious complications arise as the result of these effects. Toxic manifestations of nausea and vomiting were observed in several patients following HN2 administration, but the intensity of these symptoms varied greatly.

CONCLUSIONS

Methyl-bis-(B chloroethyl) amine is a useful drug in the treatment of some cases of inoperable bronchogenic carcinoma. This is especially true if the carcinoma is anaplastic. Whether nitrogen mustard is more effective than x-ray therapy in the treatment of bronchogenic carcinoma remains to be evaluated. Ultimately nitrogen mustard therapy, like irradiation, proves ineffective. Ease of administration, availability in communities where irradiation may not be obtainable and absence of skin reaction are advantages of nitrogen mustard as compared to roentgen-ray therapy. Methyl-bis is a useful drug, but is toxic and must be administered with care. Studies must be repeatedly made on the patient's blood to follow the state of the bone marrow.

RESUMEN

1) Se han descripto los efectos de la amino-metil-bis (B cloroetil) en 19 enfermos con cáncer broncogénico provado histológicamente.

2) Siete de los 19 están todavía vivos. Cinco de los siete vivos tienen cáncer anaplástico.

3) Se ha obtenido un efecto bueno en dos enfermos, que tienen cáncer anaplástico. Un enfermo está todavía vivo, nueve meses después de haber recibido tres serios y media de tratamiento con el nitrógeno de mostaza. El otro enfermo también está vivo, seis meses después de recibir dos serias de la misma droga. Los dos presentaron invasión del mediastino superior, el cual decreció en tamaño después del tratamiento.

4) El nitrógeno de mostaza generalmente produce una disminución de los glóbulos blancos y plaquetas, pero ningún enfermo ha sufrido serias complicaciones como consecuencia del mismo. Se han observado manifestaciones tóxicas, como ser nauseas y vómitos, en varios enfermos, después de la administración de la droga, la intensidad de estos síntomas, varía grandemente.
CONCLUSIONES

Metil-bis-(B cloroetil) amino es una droga útil, en el tratamiento de algunos casos inoperables de cáncer broncopénico. Esto es especialmente cierto en el caso de cáncer anaplásico. Todavía no se ha podido evaluar si el tratamiento con esta droga, es más efectivo que el tratamiento con los rayos X, en el cáncer broncopénico. Finalmente, esta droga como los rayos X, no son efectivos. Esta droga presenta algunas ventajas sobre el tratamiento con rayos X, como ser fácil administración, fácil de obtener en lugares en los cuales no hay rayos X y la ausencia de lesiones de la piel. Esta droga es útil, pero es tóxica y debe ser administrada con cuidado. Deben hacerse análisis repetidos de sangre de los enfermos, para seguir de cerca el estado de la médula ósea.

REFERENCES


