IMPOTENCE
AFTER RECOVERY FROM
GUILLAIN BARRÉ SYNDROME

FOR MEN RECOVERING FROM GUILLAIN BARRÉ SYNDROME, THERE HAS BEEN A SIGNIFICANT INCREASE IN IMPOTENCE COMPARED TO THE GENERAL MALE POPULATION. THE CAUSE IS RELATED TO INCOMPLETE REPAIR OF AUTONOMIC NERVE FIBERS SUPPLYING THE PELVIC AREA AS PROVED IN A RECENT SURVEY OF ALMOST 4M MEN, Kopel Burk, MD; Alan Weiss, PhD

Guillain Barré syndrome (GBS) is a relatively rare paralytic neurological disease, affecting 1 to 2 people per 100,000 in the population yearly. The mortality rate is between 1 and 5.6 percent. Recovery occurs in 85 percent of patients within 6 months, though improvement may continue for as long as 24 months. Less than 10 percent of patients are left with severe residual disabilities.

Only an occasional article mentions impotence in the current literature. Ropper's article in 1992 discussed the problem, suggesting that impotence following GBS most likely would be found in those men with severe dysautonomia and sensory loss during the acute phase of GBS, or in men suffering from depression.

The current study was designed to learn what effect GBS had on impotence following recovery. If the incidence of impotence was significant in our surveyed male population, we hoped to determine if the increased incidence correlated with the severity of the residual disability and whether depression or other medical conditions were contributing factors.

MATERIALS AND METHODS

With the help of the GBS Foundation International, 10,000 patients in the United States and England received a questionnaire. These surveys were distributed at GBS support group meetings or through The Communicator, the Foundation's official publication, from May 1994 through June 1995. There were 847 replies; 27 were discarded because the gender of the respondent could not be identified; 4 were discarded because the questionnaires were partially filled out by a parent for a young child; 62 were discarded because they were received after the database had been entered into the computer. Of 754 usable replies, 396 were from men and 358 from women. This article concerns the 396 male respondents.
RESULTS

Table 1 displays the data concerning impotence in the surveyed population of 396 men. The incidence of impotence following recovery from GBS was 42 percent (166 of the 396 men). Prior to GBS, only 35 patients (8.8 percent) were impotent. Table 1 also compares our results with the Kinsey statistics.

Table 1. Impotence following GBS; A Comparison with Kinsey’s statistics.

<table>
<thead>
<tr>
<th></th>
<th>All Men</th>
<th>Men 20-40</th>
<th>Men 41-55</th>
<th>Men 56-64</th>
<th>Men 66+</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population</td>
<td>396</td>
<td>66</td>
<td>109</td>
<td>99</td>
<td>122</td>
</tr>
<tr>
<td>Impotence</td>
<td>166(42%)</td>
<td>11(17%)</td>
<td>30(28%)</td>
<td>51(52%)</td>
<td>74(61%)</td>
</tr>
<tr>
<td>[Kinsey 2%]</td>
<td>[Kinsey 7.7%]</td>
<td>[Kinsey 25%]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Impotence before GBS</td>
<td>35(8.8%)</td>
<td>1(1%)</td>
<td>2(2%)</td>
<td>8(8%)</td>
<td>24(20%)</td>
</tr>
<tr>
<td>Still try to make love</td>
<td>122(73%)</td>
<td>7(64%)</td>
<td>25(83%)</td>
<td>35(69%)</td>
<td>55(74%)</td>
</tr>
</tbody>
</table>

In the 20-40 year-old age group, 17 percent (11 of 66 men) reported problems with impotence following GBS. Prior to GBS, only 1 man (1 percent) reported erectile dysfunction. This difference is significant, P=0.002. In the Kinsey report, 10 of 513 men (1.9 percent) in the 20-40 year-old age group of the general I group is significant, P=0.000002.

For men ages 41-55 who had GBS, the incidence of erectile dysfunction was 28 percent (30 of 109 men). Prior to GBS, 2 men (2 percent) were impotent, a significant difference, P=2x10. In the Kinsey population ages 41-55, 9 of 1,335 men (6.7 percent) were impotent. The difference between the 2 per-cent rate of impotence prior to GBS and the Kinsey rate of 6.7 Percent is statistically insignificant (0.05). However, the 28 percent rate of impotence after CBS is significant.

The incidence of erectile dysfunction in the 56-65 year-old group following recovery from GBS was 52 percent (51 of 99 men). Prior to GBS, 8 (1 percent) were impotent. This change is significant, P=10. For the Kinsey group be-tween the ages of 60 and 65, the rate of impotence was 25 percent. The difference between our post GBS group and the Kinsey group is significant, P=0.002.

In our younger population ages 20 to 55 years, the rate of impotence was 23 percent (41 Of 175 men). Kinsey reported that 9 of 134 men between the ages of 50 to 55 years were impotent, a rate of 6.7 percent. Using the Fisher exact test, the probability that our population was
statistically the same as the Kinsey population is extremely small (P=0.0001). Our population was younger than the Kinsey group, but its incidence of impotence was significantly higher.

A large number of men were over 65 years of age, ranging from 66 to over 80 years old. Of 122 men in this group, 74 (61 percent) were impotent. Prior to GBS, their rate of impotence was 20 percent (24 of 122). This change is significant, P=10.

We could not compare the 66 and older population with the Kinsey statistics since we did not break down this large group by age. Table 1 shows that the majority of men in the study reporting problems with impotence had sexual interest and desire and were involved in some form of sexual activity. This finding will be discussed later.

Table 2 displays the relationship between the degree of residual disability and the incidence of impotence in age groups below 56, 56-65, and over 65. Table 2 shows that the greater the residual physical disability, the greater is the incidence of impotence. In patients 55 years and younger there was no difference in the rate of impotence between men who had recovered completely and those who reported only mild residual disability following GBS. In this same age group, those with moderate residual disability and erectile dysfunction were statistically indistinguishable from the men with impotence who reported severe residual physical disability. The difference between the "no/mild" and the "moderate/severe" groups was significant (P=.00002).

<table>
<thead>
<tr>
<th></th>
<th>All Men</th>
<th>Men ≤ 55</th>
<th>Men 56-64</th>
<th>Men 66+</th>
</tr>
</thead>
<tbody>
<tr>
<td>No residual Impotence</td>
<td>41 (24%)</td>
<td>23 (13%)</td>
<td>7 (14%)</td>
<td>11 (55%)</td>
</tr>
<tr>
<td>Mild Impotence</td>
<td>176 (32%)</td>
<td>91 (13%)</td>
<td>42 (45%)</td>
<td>43 (58%)</td>
</tr>
<tr>
<td>Moderate Impotence</td>
<td>128 (53%)</td>
<td>41 (13%)</td>
<td>33 (55%)</td>
<td>54 (61%)</td>
</tr>
<tr>
<td>Severe Impotence</td>
<td>51 (63%)</td>
<td>20 (45%)</td>
<td>17 (76%)</td>
<td>14 (71%)</td>
</tr>
</tbody>
</table>

**DISCUSSION**

Impotence, or erectile dysfunction, is defined as the inability to achieve and maintain a penile erection sufficiently rigid to permit satisfactory sexual intercourse. Although other sexual functions also may be abnormal, impotence is not synonymous with the loss of sexual interest or desire, or the ability to ejaculate or to have an orgasm. Erectile dysfunction is considered a common condition in men. It is estimated that 10 percent of men over the age of 21 are
impotent. The problem may affect as many as 10 to 20 million American men. Though an insignificant finding in the younger male population (0.1 percent at age 20 up to 2.6 percent at age 45), it becomes an important problem by age 60 when the incidence of impotence increases to 18.4 percent. At age 65, impotence affects one-fourth of the male population, and by age 75 over one-half of all men are impotent. Although a more recent study cites a somewhat higher incidence of erectile dysfunction of 5 percent at age 40, the rest of the statistics remain about the same as in the Kinsey report.

The majority of men who have sexual dysfunction have an organic basis for their impotence – vascular, neurogenic, or hormonal. In GBS, autonomic nervous system dysfunction often is present, manifested by urinary retention, gastric atony, constipation, problems with sweating and body temperature regulation, loss of visual accommodation, and impotence. In its more severe form, marked changes in blood pressure may be present, and even death may occur from cardiac arrhythmias. Recovery is gradual and may be incomplete.

Most nerve fibers of the sympathetic and parasympathetic nervous system are made up of small (2-6 microns) myeliated or unmyelinated fibers. Since GBS is an acute demyelinating disease that also may involve the axon, involvement of these fibers causes the dysautonomia seen in this illness. With recovery, repair of the myelin sheath and regeneration of the axon may be incomplete. We feel that this incomplete repair is a reasonable explanation for the significant incidence of impotence found in our surveyed population. It is interesting to note that among the 41 men who felt they had recovered completely, the incidence of impotence was higher than in the general population of the same age group. In addition, 5 men reported that their reflexes had not returned (12 percent). While testing for reflexes and measuring skeletal muscle strength is easy to quantify, evaluating subtle abnormalities of the pelvic autonomic nervous system is more difficult.

The data suggest that the reported high rate of impotence in GBS is organic in etiology. This conclusion relies on the above pathologic findings as well as on somewhat indirect argument. First is the recognition that the majority of men who are impotent have an organic basis for their impotence.

In the survey, we questioned patients who were also being treated for hypertension, heart disease, and chronic lung disease. We found no correlation between these illnesses, or medication used to treat these illnesses, and the reported increased incidence of impotence. In the survey, we questioned patients who were also being treated for hypertension, heart disease, and chronic lung disease. We found no correlation between these illnesses, or medication used to treat these illnesses, and the reported increased incidence of impotence following “recovery” from GBS. Of 52 men with heart disease, 26 were impotent. From our sample in that age distribution, we expected to find 28 men who were impotent. Eighty-three men were on medication for hypertension; 40 of whom were impotent. In the age distribution of our sample, we expected to find 41 men who were impotent. Therefore, we found no increase in the rate of impotence in patients with heart disease or in our men with hypertension who were on medication.
There were only a few men with chronic lung disease. Most were impotent, but the patient population was too small to evaluate statistically.

The only medical condition that seemed associated with impotence was diabetes. There were 18 males with diabetes, and though the number was too small to be statistically significant, 50 percent were impotent following GBS.

None of our respondents reported being depressed or taking medications for depression. The majority of the men who were impotent enjoyed going out socially, and most who were impotent still had sexual interest and desire and still tried "to make love."

In psychiatric and urologic literature, investigators described an interesting clinical observation that helps separate a psychological from an organic cause of impotence. The majority of men who had an organic etiology for impotence still had sexual interests and desires and still tried to "make love." Those whose impotence was psychogenic, generally lost interest in sex and sexual activity.

Our data, therefore, points to an organic cause for impotence following recovery from GBS. We have suggested that the incomplete repair of damaged autonomic nervous system fibers in the pelvic area is the etiology.

The problems inherent in this study are similar to the problems of all such mail surveys. Participation is purely voluntary, so the results may not apply to the general population of men who have had GBS. However, information about sexuality or sexual function following an illness or injury cannot be obtained by reviewing the records of patients while they are still in an acute care hospital, or even when they are in a convalescent or rehabilitation center. Information regarding sex and sexual function can only be validated once patients have recovered enough to return to the community. A mailed anonymous survey sent after recovery, or when the patient is in a stable state, is the only practical way to gather this information.

The terms mild, moderate, and severe were not defined in the questionnaire. Most of the men who had GBS in our survey were associated with the GBS Foundation. Many had been to support groups or had attended one of the GBS symposia and, therefore, we believe had a reasonable understanding regarding the full range of disabilities that may remain following GBS.

Patients were asked to assess their own level of disability. The data show that in men 55 and under there were only two categories relative to impotence, those who had "no/mild residual" and those who had "moderate/severe residual" disability. Given these two extremes, we believe that most men placed themselves in the correct category.

GBS literature does not separate male from female when reporting the percentage of patients left with severe residual disability. In the 754 patients in our initial GBS population, there was
reported a 10 percent incidence of severe residual disability, which is in line with the general literature. Our male population had a 13 percent incidence of severe residual and our female population reported only an 8 percent incidence. Perhaps future studies of large GBS populations should be reported by gender.

CONCLUSION

The results of our study of 396 men surveyed more than three years after their acute illness with GBS show a significant increase in the rate of erectile dysfunction compared to the general male population of the same age groups. Our research strongly suggests that the reported impotence is organic and related to the residual autonomic dysfunction following GBS. Furthermore, the impotence seems directly related to the severity of the residual disability.

References are available upon request.

*Dr. Burk is in Private Practice in Millburn, NJ. Dr. Weiss is a member of the technical staff at Bell Lab.*

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Some persons who received the 1976-77 swine flu injections developed Guillain Barré Syndrome. This experience has prompted concerns about the safety of influenza and other immunizations in patients who have had GBS (Guillain-Barré Syndrome) or CIDP (Chronic Inflammatory Demyelinating Polyneuropathy). Since the 1976-77 experience, other published studies have not found a clear increase in risk for GBS among influenza vaccine recipients. At this point, the association of GBS and influenza vaccines subsequent to the 1976 swine flu vaccine remains uncertain. Nevertheless, GBS patients often raise concerns about the safety of vaccines. Based upon the medical literature, the following guidelines on the subject can be offered.

- The risk of developing GBS from most vaccines is very small. Even during the 1976 swine flu vaccination campaign, the increase in risk was about 1 case per 100,000 vaccinated persons. In most patients, the benefits of vaccines far outweigh the small chance of a complication.

- There have been rare reports of patients developing GBS and other nervous system disorders after the administration of many vaccinations. Thus, vaccines cannot be considered completely safe. However, for most immunizations, the risk of developing GBS is extremely small. Therefore, it is not usually wise for a patient to be deprived of the potential benefits of an immunization. There is an exception to this advice. The vaccine against meningitis, meningococcal diphtheria vaccine by Sanofi Pasteur, is marketed as Menactra®. Adverse event reports suggest an increased risk of GBS following vaccination with Menactra®. Accordingly, it is recommended that persons previously diagnosed with GBS should not receive Menactra® vaccine.

- In most cases where GBS developed after an immunization, proof that the injection caused the GBS has not been established. However, if a patient's GBS or CIDP was found to have been triggered by a specific immunization, then it probably should not be given again. If a patient is unsure about this, they should consult their physician.

- Information about the risks of immunizations to GBS and CIDP patients is very limited. Since vaccines are usually effective and safe, a decision not to use them warrants careful consideration.

- Ultimately, the decision about receiving an immunization is best determined by a discussion between the patient and the treating physician who can take into account each individual patient's medical history.

Joel Steinberg, M.D.

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