Psychogenic aphonia: No fixation even after a lengthy period of aphonia

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Summary

Background: Although psychogenic aphonia is considered to be a conversion disorder, aphonic patients are primarily treated symptomatically. This is because it is considered of overriding importance to elicit a voice quickly to avoid fixation of the aphonia. The aim of this study was to show that, for patients exhibiting the symptom of voicelessness, not eliciting the voice immediately will not lead to a permanent aphonia.

Methods: Between February 2000 and May 2006, aphonia was diagnosed in 22 patients. Effects of short-term psychodynamic psychotherapeutic intervention and voice therapy were studied in a follow-up of three years, on average.

Results: Twenty one patients recovered their voices; 6 even before their first medical examination, 13 after an average of 12 weeks and 2 patients after 2 and 3 years respectively. One patient who has been in psychiatric therapy for years, as a result of having suffered serious abuse, failed to regain her voice.

Conclusions: Even after a lengthy period of aphonia a complete recovery of the voice function is possible in nearly all cases. Countertransference phenomena in therapists are discussed as the possible reason why they usually decide on treatment aimed primarily at dealing with the symptoms.

Key words: psychogenic aphonia; conversion disorder; symptom-orientated therapy; short-term dynamic psychotherapy

Introduction

The disease pattern

“Psychogenic aphonia”, formerly called “hysterical aphonia” or “acute sudden voice loss” and nowadays additionally called “functional aphonia” or “conversional aphonia” is a rather rare disorder with a point prevalence of 0.4% [1–3]. It appears about 8 times more frequently in females than in males. Although a hyperfunctional type (a strong contraction of the vocal cords) does exist, the usual form is the hypofunctional type: as soon as the patient tries a deliberate phonation, the vocal folds approximate, but remain open. The patient whistles only “either entirely without sound or with short insertions of extremely high phonations which sound breathy or strained. Resonant coughing can nearly always be produced” [4]. Psychogenic aphonia is traditionally regarded as a conversion disorder [5, 6] and classified accordingly in the ICD-10 under “Dissociative Disorders” (F44) as a “Dissociative Motor Disorder” (F44.4), and in the DSM-IV under “Somatoform Disorders” as a “Conversion Disorder” (300.11).

Traditional therapeutic interventions

Despite this explicit psychiatric classification, the aim of the primary intervention has traditionally been to reestablish voice production in a single session with a treatment lasting between a few seconds up to several hours [7, 8] or in just a few sessions. This used to be done by taking the patient by surprise with brute force using methods like “Muck’s ball” [9] (see fig. 1), a sudden obstruction of the entrance to the larynx “until a sensation of suffocation produced a reflex cry of alarm” [10], “Reichert’s hook”, electrical pulses, the application of “an electromagnet to the tongue, “Flateau’s manoeuvre” (grasping the tongue and larynx with both hands), irritation by dripping water, blowing powder into the larynx or by brushing the mucosa with cocaine.

Over time the techniques became more sophisticated, for example initiating the voice by coughing or clearing the throat which in these patients is nearly always voiced. The self-imposed limitations in treatment, however, remained. The recovery of the voice was defined as success in itself, although the psychosocial background which led to the aphonia remained the same.
tional dysphonia seems to be a gradual one [12–
15]. Rubin [16] promoted a new, general “clinician-friendly psychodynamic model”, and regarding psychogenic aphonia which differentiates between 3 types of conversion reaction: the classical Freudian conversion, a cognitive-behavioural-conversion (in which no unconscious conflicts have to exist) and a “psychogenic-habituated conversion” (in which psychogenic conflicts have largely been solved). Baker [15] described Butcher’s model of cognitive-behavioural-conversion as “the most comprehensive and relevant theoretical explanation to date” and suggested, in addition, using a concept proposed by Lane [17] which asserts that patients suffering from FVD, especially those with psychogenic aphonia, function on a lower level of emotional awareness. These two theoretical approaches provide an excellent justification for supplementing symptom-orientated voice therapies with the Cognitive-Behavioural Therapy (CBT) [13, 14, 18]. The disadvantage of this strategy, however, is that essential psychodynamic-systemic aspects will get lost: the impact of early attachments, unconscious anxieties, relationship conflicts, and primary or secondary morbid gain will not be given adequate consideration. The aphonia, the symptom, is still regarded as a malady which has to be combated, and is the focus of therapeutic efforts. In behaviour therapies, suggestive techniques are still being used to reestablish the aphony voice [19]. In the treatment of “psychogenic-habituated conversion”, a firm authoritative approach by the speech therapist is thought to be essential (Butcher 2007). The aim is “to physically, reset the mechanism” of the voice [16] by means of voice therapy, or possibly in the future, by a repetitive transcranial magnetic stimulation (rTMS) to the right motor cortex [20]. The main objective of voice-therapy in many places still remains to be the recovery of the voice during the first day of vocal exercises” [21]. However, giving priority to dealing with the symptom contradicts psychodynamic insights about the emotionally stabilising function of symptoms.

The ostensible reason for the discrepancy between understanding and treatment

The main argument for focusing therapy, so decisively, on correcting the symptoms is based on a supposed serious risk: that the aphony will become permanent if the patient gets used to it. The “immediate” initiation of therapeutic intervention using exercises is thought to be very important “because with every day that the aphony continues the disorder is likely to become more and more fixed” [8, 10, 22]. However, there is a lack of studies in the literature reporting fixation of the aphony after a lengthy period of voicelessness. Only in old reports, when the therapy of choice was hypnosis, can relevant information be found. However this tends to show the opposite picture. In 1887, Bottey reported a woman who was “cured” of an aphonia lasting 18 months in 5 sessions of hypnosis [23]. Of the 5 aphony women who had been aided by Schnitzler to get their voices back by hypnosis, one woman had been aphony for 2 years before the treatment started, another woman for one year and 3 more women for 2 to 3 months [23].

Objectives of the study

Since the cause of the aphony is psychogenic, this implies that failure to induce phonation in a patient after a lengthy period of voicelessness will not cause the aphony to become fixed if s/he is supported by suitable medical advice, by not only cognitive-behavioural therapy but also by psychodynamic-systemic psychotherapeutic intervention, and by logopedic therapy with a bio-psycho-social basis.

A psychodynamic-systemic approach helps the patients to understand which elements in their life history have given rise to the interpersonal conflicts that have brought them to the helplessness of aphony. When they are emotionally more stable and more capable of acting, the patients themselves will define the moment when they speak with voice again. In this study, it will be shown that this approach of respecting the symptom will not lead to a fixation of the aphony.
Method

Between February 2000 and May 2006, psychogenic aphonia was diagnosed in 22 patients, 19 females (mean age 41 years, standard deviation [SD] 15 years) and 3 males (mean age 34 years, SD 21 years), at the Division of Phoniatrics of the Department of Oto-Rhino-Laryngology, Head and Neck Surgery of Bern University Hospital. Videolaryngoscopy excluded organic findings and normal mucosa of the vocal folds was documented (70° and 90° rigid laryngoscope, Karl Storz, Tuttlingen, Germany; RP-Szene Videodocumentation, Rehder and Partner, Hamburg, Germany). The diagnosis was confirmed by a lack of adduction of the vocal folds during phonation, but normal movement and complete closure of the vocal folds during coughing.

“Fixation of aphonia” is pragmatically defined as persisting voicelessness for more than one year after confirmation of the diagnosis.

Six patients were no longer completely aphonic at the first consultation, but could speak again with a very weak and breathy voice. However, the diagnosis could be confirmed unambiguously on the basis of the patients’ history and also on the discrepancy between the normal aspect of the larynx on the one hand and the severe dysphonia with reduced adduction of the vocal folds during phonation and the complete closure during coughing on the other.

Fourteen patients (64%) had previously experienced aphonic phases (on average 6 phases) lasting from a few days to up to 3 years. The time between the beginning of the current aphonia and the consultation at our department was, on average, 20 days (SD 18 days). One exceptional case was excluded from this calculation, whose aphonia had already lasted for more than one year by the day of consultation. Detailed information on the patients is provided in table 1.

The psychogenic genesis of the disorder was explained to the patients with the aid of the videolaryngoscopic findings. Psychodynamic-systemic psychotherapeutic intervention and voice therapy was suggested to each patient.

Table 1

Patients with psychogenic aphonia (n = 22). Data concerning voice and therapy success.

<table>
<thead>
<tr>
<th>Consecutive Pat. No</th>
<th>Sex</th>
<th>Age</th>
<th>No. of earlier aphonic phases</th>
<th>Longest duration of earlier aphonic phases (weeks)</th>
<th>Voice at first consultation</th>
<th>Duration of the current aphonia (days)</th>
<th>Therapy and epicrisis</th>
<th>Recovery of the voice after (weeks)</th>
<th>Success of psychotherapy</th>
<th>Relapse (no. of new aphonic phases)</th>
<th>No relapse for (years)</th>
<th>Group</th>
</tr>
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<tr>
<td>6 F 63</td>
<td>10</td>
<td>0.6</td>
<td>dysphonic</td>
<td>5</td>
<td></td>
<td></td>
<td>12</td>
<td>3 initially</td>
<td>no</td>
<td>0</td>
<td>5.6</td>
<td>A</td>
</tr>
<tr>
<td>8 F 48</td>
<td>5</td>
<td>0.8</td>
<td>dysphonic</td>
<td>6</td>
<td></td>
<td></td>
<td>7</td>
<td>12 initially</td>
<td>yes</td>
<td>3</td>
<td>4.7</td>
<td>A</td>
</tr>
<tr>
<td>14 F 32</td>
<td>8</td>
<td>1.0</td>
<td>dysphonic</td>
<td>3</td>
<td></td>
<td></td>
<td>no</td>
<td>8 initially</td>
<td>yes</td>
<td>2</td>
<td>1.6</td>
<td>A</td>
</tr>
<tr>
<td>16 F 41</td>
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<td>1.0</td>
<td>dysphonic</td>
<td>4</td>
<td></td>
<td></td>
<td>12</td>
<td>1 initially</td>
<td>no</td>
<td>7</td>
<td>0.5</td>
<td>A</td>
</tr>
<tr>
<td>17 F 41</td>
<td>5</td>
<td>0.7</td>
<td>dysphonic</td>
<td>4</td>
<td></td>
<td></td>
<td>suggested</td>
<td>5 initially</td>
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<td>0</td>
<td>2.1</td>
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<td>19 F 55</td>
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<td>dysphonic</td>
<td>14</td>
<td></td>
<td></td>
<td>refused</td>
<td>ongoing external therapy</td>
<td>initially</td>
<td>partially</td>
<td>1.9</td>
<td>A</td>
</tr>
<tr>
<td>1 F 57</td>
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<td>aphonic</td>
<td>14</td>
<td></td>
<td></td>
<td>11</td>
<td>1 (aborted)</td>
<td>22.0</td>
<td>no</td>
<td>6.5</td>
<td>B</td>
</tr>
<tr>
<td>4 F 45</td>
<td>0</td>
<td>aphonic</td>
<td>14</td>
<td></td>
<td></td>
<td></td>
<td>9</td>
<td>refused</td>
<td>9.0</td>
<td>0</td>
<td>6.3</td>
<td>B</td>
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<tr>
<td>5 F 38</td>
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<td>56</td>
<td></td>
<td></td>
<td></td>
<td>6</td>
<td>7</td>
<td>9.0</td>
<td>yes</td>
<td>5.8</td>
<td>B</td>
</tr>
<tr>
<td>7 F 68</td>
<td>25</td>
<td>7.0</td>
<td>aphonic</td>
<td>63</td>
<td></td>
<td></td>
<td>17</td>
<td>4</td>
<td>34.0</td>
<td>partially</td>
<td>4.4</td>
<td>B</td>
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<tr>
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<td>0</td>
<td>aphonic</td>
<td>14</td>
<td></td>
<td></td>
<td></td>
<td>31</td>
<td>5 (counseling of parents)</td>
<td>13.0</td>
<td>yes</td>
<td>3.6</td>
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<tr>
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<td>0</td>
<td>aphonic</td>
<td>42</td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>(aborted) 10</td>
<td>15.0</td>
<td>yes</td>
<td>3.6</td>
<td>B</td>
</tr>
<tr>
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<td>28</td>
<td></td>
<td></td>
<td></td>
<td>7</td>
<td>9</td>
<td>7.0</td>
<td>yes</td>
<td>3.6</td>
<td>B</td>
</tr>
<tr>
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<td>3.0</td>
<td>aphonic</td>
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<td></td>
<td></td>
<td>30</td>
<td>39</td>
<td>17.0</td>
<td>yes</td>
<td>3.0</td>
<td>B</td>
</tr>
<tr>
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<td>aphonic</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td>11</td>
<td>3</td>
<td>0.7</td>
<td>partially</td>
<td>3.1</td>
<td>B</td>
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<tr>
<td>18 F 14</td>
<td>0</td>
<td>aphonic</td>
<td>15</td>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>(aborted) 1</td>
<td>17.0</td>
<td>no</td>
<td>1.6</td>
<td>B</td>
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<tr>
<td>20 F 25</td>
<td>3</td>
<td>1.4</td>
<td>aphonic</td>
<td>3</td>
<td></td>
<td></td>
<td>6</td>
<td>6</td>
<td>0.2</td>
<td>yes</td>
<td>1.8</td>
<td>B</td>
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<tr>
<td>21 F 45</td>
<td>6</td>
<td>2.0</td>
<td>aphonic</td>
<td>35</td>
<td></td>
<td></td>
<td>14</td>
<td>6</td>
<td>5.0</td>
<td>partially</td>
<td>0.5</td>
<td>B</td>
</tr>
<tr>
<td>22 M 38</td>
<td>3</td>
<td>0.4</td>
<td>aphonic</td>
<td>5</td>
<td></td>
<td></td>
<td>refused</td>
<td>2 (aborted)</td>
<td>0.2</td>
<td>partially</td>
<td>1.6</td>
<td>B</td>
</tr>
<tr>
<td>2 F 61</td>
<td>3</td>
<td>136.0</td>
<td>aphonic</td>
<td>14</td>
<td></td>
<td></td>
<td>18+IPT</td>
<td>3+IPT (5)</td>
<td>183.0</td>
<td>partially</td>
<td>3.3</td>
<td>C</td>
</tr>
<tr>
<td>3 F 37</td>
<td>2</td>
<td>1.4</td>
<td>aphonic</td>
<td>21</td>
<td></td>
<td></td>
<td>65+IPT</td>
<td>3+IPT (5)</td>
<td>100.0</td>
<td>no</td>
<td>4.8</td>
<td>C</td>
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<tr>
<td>15 F 20</td>
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<td>aphonic</td>
<td>18 months</td>
<td></td>
<td></td>
<td></td>
<td>refused</td>
<td>ongoing external therapy</td>
<td>no</td>
<td>unknown</td>
<td>aphonie</td>
<td>D</td>
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</table>
The psychotherapeutic intervention, in the form of a short-term dynamic psychotherapy, includes a reflection of the biographical history of attachment, an analysis of current relationship conflicts, a raising of the “level of emotional awareness” [15, 24] by an empathetic dialogue about everyday situations (not by exercises) and working on the patients’ conflict over speaking out (CSO [25]).

The voice therapy on a bio-psycho-social basis included sensory awareness, relaxation, breathing, and voice exercises in precise accordance with the needs and wishes of the patients. Psychotherapy and voice therapy started simultaneously. Neither at the time of diagnosis nor during the subsequent therapy were the patients forced to produce phonation.

**Results**

To analyze the therapeutic effects of the interventions and to determine how well-founded it is to expect the aphonia to become permanent if the symptom is not treated immediately, it seems reasonable to divide the 22 patients into the following four groups:

**A** (n = 6): Patients who could already speak again with a voice at the time of the initial consultation; therefore those who were no longer aphonic but dysphonic.

**B** (n = 13): Patients who were aphonic at the initial consultation, but recovered their voices within one year with non-directive voice therapy and short-term dynamic psychotherapy.

**C** (n = 2): Patients who took more than one year before they were able to speak with a voice despite intensive therapy.

**D** (n = 1): A patient who had been aphonic for a long time and remained voiceless.

(For details refer to table 1.)

Patients of **group A** (n = 6), those who could already speak with a voice at the time of the initial consultation, are characterized by having experienced previous aphoncic phases (on average 6–7) of fairly short duration (5 days to 2 weeks). They requested help soon after the beginning of the aphonia (within 6 days on average). In this group, there is still a likelihood of further relapses but very little risk that the voicelessness will become fixed. Indeed, 3 of these patients (50%) had suffered relapses (2–7 phases of aphonia) between the initial consultation and the cut-off date of the study (20.12.2007), but on average had been relapse-free for 2.7 years (SD = 2.0 years).

Patients of **group B** (n = 13) had been aphonic, at the time of the initial consultation, for, on average, 26 days (SD = 19.1). After a mean 7.8 sessions of psychotherapy (SD = 10.3) and 12 units of voice therapy (SD = 9.6), each patient had recovered a speaking voice after 11.5 weeks (SD = 9.7) on average. The subjective assessment of the psychologist was used as a discreet way of measuring the success of the psychotherapeutic intervention. “Successful” means that the patient had recognized the psychosomatic context and had initiated psychosocial changes. “Partially successful” means that the patient had only recognized the psychosomatic context. Measured in this way, the results were: 6 “successful” (46%), 4 “partially successful” (31%), 2 “not successful” (15%). One patient refused psychotherapeutic intervention and 3 patients gave up the therapy before completion. In the following years, one patient suffered 3 relapses, but the other patients remained relapse-free. At the end of the follow-up period the patients of this group had had their voices for on average of 3.5 years.

The 2 patients who got their voices back only after 2–3 years (group C) were females who had been through a lengthy saga of therapies before they recovered the ability to speak with a clear voice, after an unspectacular but existentially important event. One patient (No. 2) aged 60 had suffered 4 episodes of aphonia, of which the longest had lasted 3 years, when she once again became aphonic. After many attempts at therapy, phoniatric, logopedic, psychiatric-psychotherapeutic and homeopathic treatments, including in-patient therapy at a voice treatment centre abroad, she still remained voiceless. It was only during a Yoga-exercise that she recovered her voice and for 3.3 years since then has spoken with a clear voice.

The second patient (No. 3) was a married woman aged 37 years with two children who initially denied any psychosocial conflicts, but abruptly broke up with her husband. She went from therapy to therapy including two in-patient therapies at a voice treatment centre abroad. Exactly one day before she would have lost her job because of the long duration of her voice disorder, she recovered her voice and it has remained (for 4.8 years).

The patient who remained voiceless, even after many years (group D), was a 20 year old woman (No. 17) who at the time of the initial consultation had been suffering from aphonia for 1.5 years. She had lost her voice on the occasion of a retraumatization (sexual abuse), had been treated by a number of psychiatrists, spent 4 months in a psychiatric clinic after attempted suicide, and is unable to work.
Discussion

An optimal design to evaluate the topic raised in this study would be a randomized comparison of 2 large groups of patients, of which one group would be treated by an immediate correction of the symptom, and the second group by a psychodynamic-systemic method. However, due to the relatively low incidence of psychogenic aphonia and the fact that post-therapy observation continues for several years, it would take 20 years to conduct such a study in a medium-sized medical centre. For this reason, the study is limited to a retrospective design.

The division into the 4 subgroups was performed because patients who were able to speak with their voice at the time when the diagnosis was established, could not be included in the evaluation of the duration of the aphonia. It would also have been confusing to treat the data of patients who had been suffering from aphonia for several years at the time of diagnosis, statistically in the same manner as the other patients.

No evidence was found in the data of any of the four subgroups suggesting that failure to induce immediate phonation had led to fixation of the aphonia. It is indeed possible that pressure for quick phonation would have allowed the patients to return sooner to their familiar social roles. However, this would, in fact, have been a dubious help, if it is presumed that psychogenic aphonia basically arises from a wish to change unsatisfactory social roles. The continuation of the aphony during the initial stages of the therapy and the social withdrawal often connected with it, may indeed be experienced as painful, but they provide a basis for patients to rethink their personal situation and are a starting point for changing ingrained patterns of social contact.

The results of this study show that the concern that each additional day of voicelessness further consolidates the psychogenic aphonia, up to the point where it can no longer be treated by therapy, is unfounded. The techniques using surprise or suggestion to provoke voice production are fundamentally incompatible with the psychogenic nature of the dysfunction [26].

The high “success” rate with inducing phonation in the very first consultation – Maniecka-Aleksandrowicz [21] for example, reports success in 82% of the patients – is deceptive. Some patients who produce an almost normal voice in the first consultation have probably come to the doctor ready to give up a symptom which has ceased to be helpful, or because they didn’t want to let it go by themselves [27]. However, for many others, who are not yet ready for this step, the suggestive therapeutic approach simply removes the symptom or even snatches it away. They are still stuck with their psychosocial conflicts and still strongly need the symptom for their mental balance. The precision of the unconscious timing of one patient from group C, who got her voice back on the very day before she would have lost her job, is striking and provides additional evidence that the moment at which the voice is recovered has to be determined by the patients themselves.

The only patient in subgroup D seems to need her voicelessness along with other withdrawal and safety mechanisms for longer, in the same way that other severely traumatized patients need their somatizations.

The technique of correcting the symptom ignores the stress which may arise when the patient loses his symptom, this in turn leading to a possible increase in both his anxieties and defence mechanisms. Furthermore, the correction of the symptom sends a false message, namely that: “The voice loss is much more important than all of the psychosocial suffering underlying it.”

An explanation for the common directive and mechanistic way of dealing with aphonic patients may lie in the possibility that the therapists are, indeed to some extent, aware of the difficult mental background behind the disorder but – perhaps out of uncertainty – choose not to go into it. However, another aspect also seems to play an important role. From studying the literature on psychogenic aphonia the impression is given, emphasized by Gutowski and Dittmann [28], that the symptom itself or the patient who comes in for treatment seem to be rather burdensome. This burden is a problem of transference and countertransference. The doctor-patient-relationship reflects the same difficulties which the patient usually encounters with other human beings, and unless the therapist pays careful attention to his own emotional reactions, he is in danger of reacting unconsciously in exactly the same way that fits the pattern of experience constantly provoked by the patient: treating him/her as a victim and everyone else as culprits.

Gerritsma [29] indicates the way in which this happens: the silence and outward amenability of the aphonic patient, at first, makes the therapist feel helpless and, then, as the therapist attempts to cope with this, inspires either aggressive or extremely protective impulses. Voicelessness gives the aphonic patient so much power over his/her social environment that even professional helpers tend to resort to countervailing power in their attempt to overcome their helplessness. They try to fight the power of voicelessness, and this stops them being able to deal with the patient in a helpful way. That is why after diagnosis the clinician needs to become a “temporary supporting psychotherapist” who – as Aronson [27] describes this important role – encourages the patient to speak about the circumstances and experiences surrounding the loss of his/her voice. From the start the therapist should show an interest in the whole life situation of the patient (not only in the loss of his/her voice) and explain, not all the time but now and then, how helpful it could be for the patient to accept psychotherapeutic assistance.

This kind of conversation can take place in one or more therapeutic sessions, perhaps until
the patient feels ready to attempt a session with a psychotherapist. In order to fulfill such a role, the clinician must resist a whole set of temptations. Although he sees himself primarily as an organic therapist who has access to a range of machine-aided diagnoses he has to be careful not to resort to inspecting the larynx as soon the conversation becomes difficult (in moments when he doesn’t know what to say next). This is especially important when the patient demands objective investigation. Furthermore the therapist has to find ways not to give in to a number of expectations. On the conscious level, which can therefore be articulated, the patient expects him to restore his/her voice as soon as possible. His/her closest associates impatiently expect the same. The referring physician too expects speedily rather than deep-rooted success. If voice therapy and psychotherapy can start at the same time, this creates less of an impression that these expectations are being ignored.

Conclusions

The concern that patients with psychogenic aphonias are not immediately induced to produce vocalization will develop a fixation of the aphonia, is not substantiated. Rather, diagnostic treatment should be guided by psychotherapeutic thinking.

“...I believe that diseases are keys which can open certain gates for us. I believe there exist certain gates which only disease can open.” (André Gide)

References


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