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Physiology of Psychogenic Movement Disorders

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Abstract

Psychogenic movement disorders (PMDs) are common, but their physiology is largely unknown. In most situations, the movement is involuntary, but in a minority, when the disorder is malingering or factitious, the patient is lying and the movement is voluntary. Physiologically, we cannot tell the difference between voluntary and involuntary. The Bereitschaftspotential (BP) is indicative of certain brain mechanisms for generating movement, and is seen with ordinarily voluntary movements, but by itself does not indicate that a movement is voluntary. There are good clinical neurophysiological methods available to determine whether myoclonus or tremor is a PMD. For example, psychogenic myoclonus generally has a BP, and psychogenic stimulus-sensitive myoclonus has a variable latency with times similar to normal reaction times. Psychogenic tremor will have variable frequency over time, be synchronous in the two arms, and might well be entrained with voluntary rhythmic movements. These facts suggest that PMDs share voluntary mechanisms for movement production. There are no definitive tests to differentiate psychogenic dystonia from organic dystonia, although one has been recently reported. Similar physiological abnormalities are seen in both groups. The question arises as to how a movement can be produced with voluntary mechanisms, but not be considered voluntary.

Keywords

Clinical neurophysiology; Conversion disorder; Dystonia; Gait; Myoclonus; Parkinsonism; Psychogenic movement disorders; Somatization; Tremor

1. Introduction

Psychogenic movement disorders (PMDs) are common. The disorder can look like any organic neurologic condition, but it has a psychiatric cause. Such conditions are frequent in medicine, and are sometimes called “medically unexplained symptoms”. In neurology, other than PMDs, there are, for example, psychogenic non-epileptic seizures, weakness or paralysis, sensory loss, blindness, and aphonia. PMDs are valuable to investigate because movement can be measured objectively. Originally well described by Charcot and Freud, these disorders disappeared from both neurology and psychiatry textbooks for decades. However, the patients have continued to present with these disorders and recently there has been increased interest in this topic. [1-6] From the point of view of making a diagnosis and understanding the physiology, clinical neurophysiological investigations are proving useful.

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Although we do not really understand the physiology of PMDs, the patients fall into several psychiatric categories. Somatoform disorders include conversion and somatization; somatization is different from conversion just because it is more chronic and has a broader range of symptoms. In these situations the disorder is involuntary, not apparently under any conscious control. Almost all patients fall into these 2 categories. Factitious disorder and malingering describe when the disorder is voluntary, and patients are lying when they say that it is involuntary. Factitious disorder is done to fulfill a psychological need, and malingering is purposeful behavior to achieve a goal, such as to avoid work or to obtain drugs.

To separate conversion and somatization from factitious and malingering, it appears necessary to be able to identify when a person is lying. This is not easy. The standard lie detection test is a measure of autonomic features, skin conductance, heart rate and blood pressure, and is often used by clinical neurophysiologists. However, this method appears to have many false positives and negatives. Electroencephalography (EEG) and functional MRI methods are being developed: some have substantial promises from their developers;[7-10] and some have been commercialised. None, however, focus on PMDs.

Could the movement-related cortical potential (MRCP) be used to separate voluntary from involuntary movement? This potential, identified by back-averaging the EEG triggered at the start of electromyography (EMG) activity, is a slowly rising negativity for about 1 s to 2 s prior to the EMG.[11] This negativity, called the Bereitschaftspotential (BP), has 2 phases: (i) BP1, the earlier phase; and (ii) BP2, a later, more rapidly rising negativity. This potential indicates the involvement of the premotor cortex including the supplementary motor cortex (SMA) in the preparation for movement. This can be seen with voluntary movements (most of the time) and some involuntary movements. By itself, however, the BP does not indicate that a movement is voluntary.

There is substantial psychiatric co-morbidity in patients with PMDs. Anxiety, depression and personality disorders are frequent, but are not identifiable in every patient, so there is not an obligatory correlation. In some patients, there is also an organic neurologic condition or even another movement disorder. The true incidence of PMDs is not clear, but may be about 15%. This makes the study of PMDs more difficult. When a patient is diagnosed with a PMD, it is necessary to assess whether there might also be an organic condition – the neurologic exam is tricky in this circumstance since the psychogenic signs may mask the organic signs.

There are clinical rules for determining whether a movement disorder might be psychogenic. None are absolute, but they can be helpful. One may not be able to be certain unless the patient improves with psychotherapy or, as we will see subsequently, the clinical neurophysiology is positive. A sudden onset is common. The disorder itself may be paroxysmal and come and go. There can be improvement with distraction. The disorder can be highly variable in its manifestation. Some patients are highly suggestible, and will turn the movement on and off at the examiner's request, even though the movement is said to be not under the patient's voluntary control. Secret surveillance has been used to identify patients with factitious disorder or malingering.

Psychogenic myoclonus and tremor can be identified reasonably well by clinical neurophysiological techniques, and I will discuss these first. More problematic disorders are discussed later.

2. Psychogenic myoclonus

Myoclonus should be a simple, quick movement.[12] Psychogenic myoclonus may also have this appearance, but often the movement is a bit more complex, with multiple components over time. When more complex, it no longer fits the phenomenology of any of the organic myoclonus

syndromes. Psychogenic myoclonus, like organic myoclonus, may present spontaneously, be action induced and be reflex produced. One type of myoclonus is increased startle, and increased startle or startle-like movements are frequent in psychogenic states.

The latency of reflex myoclonus should be very short, so a long latency would be indicative of psychogenicity.[13] Latencies are often difficult to tell by eye, and physiology is useful, as will be described shortly. Another clue that reflex myoclonus would be psychogenic is when the reflex occurs even though the tendon hammer is stopped just short of the tendon (without hitting it) – just the visual stimulus might provoke the psychogenic movement.

The clinical neurophysiologic assessment, as with all involuntary movements,[14,15] should include an examination of: (i) the EMG burst length and EMG antagonist muscle relationships; (ii) the EEG correlate to the movement, and (iii) for reflex myoclonus, examination of the reflex latencies.[16,17]

2.1. EMG evaluation

In myoclonus that is a fragment of epilepsy, the EMG burst length is generally 30–50 ms and antagonist muscles are always synchronous. In other forms of myoclonus, the EMG burst length is longer and antagonist muscle relationships are variable. Psychogenic myoclonus falls into this latter category. Hence, epileptic myoclonus can be ruled out with this method, but non-epileptic myoclonus cannot be.

Some forms of non-epileptic myoclonus have characteristic EMG patterns, and this would help identify them as such. When viewed with fast sweep speeds, it is possible to see some fine structure in the onset latencies of the different muscles involved. For example, in startle, orbicularis oculi is the first and most consistent muscle, sometimes with an apparent double burst. This is followed by activity in the lower cranial nerve muscles and subsequently in the upper cranial nerve muscles and limb muscles.[18] In propriospinal myoclonus, the first activity is seen in the midthoracic region and subsequent muscles are activated up and down the neuraxis at latencies suggesting a relatively slow propagation in the spinal cord.[19] In this situation, however, the pattern can be mimicked voluntarily,[20] and a patient with psychogenic propriospinal myoclonus has been reported.[21]

An example of possible misidentification of psychogenic myoclonus as organic is a report of patient with myoclonus in the setting of complex regional pain syndrome.[22] A careful look at the EMG characteristics suggested that the reported myoclonus could be psychogenic.[23]

2.2. Correlation between EEG and myoclonus

Types of epileptic myoclonus have characteristic EEG correlates. The EEG correlate is obtained by back-averaging the EEG using the onset of EMG (or movement) as the fiducial point. The best known is the potential associated with epileptic myoclonus, a brief negative–positive potential about 20 ms prior to the EMG. In non-epileptic myoclonus, generally a potential is not identified. However, in psychogenic myoclonus, a normal looking BP is seen frequently (Fig. 1),[24] which indicates premotor cortex activity but not “voluntariness.” Indeed, the presence of the BP with conversion psychogenic myoclonus is one major argument against the BP being associated with voluntariness.

2.3. Reflex myoclonus latency

The physiological correlate of reflex myoclonus is called the C-reflex. In organic myoclonus syndromes the C-reflex comes from hyperexcitability of one of several long-latency reflex pathways. All these pathways produce shorter latencies than the fastest voluntary reaction times, about 40 ms to 50 ms. In psychogenic reflex myoclonus, the latencies are similar to, and

never faster than, the fastest voluntary reaction time, 100 ms or longer depending on the type of sensory stimulus.[16,17] Moreover, like voluntary reaction times, the latencies are variable. If the reaction time appears short in this situation, the reaction might be anticipatory in response to the visual stimulus of the approaching somatosensory stimulus.

These studies can be definitive as to psychogenic myoclonus in individual patients. Additionally, they shed some light on the movement disorder. The movements have all the characteristics of voluntary movement, both in their appearance and in the EEG correlate. However, the movement is not perceived as being voluntary. This demonstrates a dissociation of movement production and the sense of voluntariness that will be discussed further below. For the moment, we can also conclude that the mechanism for production of psychogenic myoclonus and for normal voluntary movement is shared. This conclusion regarding the shared mechanism is limited to the most proximal steps in movement production as indicated by the BP, and consists mainly of premotor cortex, supplementary motor cortex and primary motor cortex in the 1 s to 2 s prior to movement.

3. Psychogenic tremor

Psychogenic tremor is often highly variable both in frequency and amplitude.[25] It tends to be present in rest, posture and kinetic action. Finger tremor is unusual. The tremor can vary or cease with distraction. Probably the most useful clinical test (that can be accurately measured and quantified physiologically) is the entrainment test. In this test, the patient is asked to voluntarily tap at various frequencies with a body part unaffected by the tremor. (If all body parts show tremor, this still can be done, with voluntary tapping of one body part while monitoring the response of the “involuntary” tremor in another body part.) The tremor is entrained if the tremor takes up the frequency of the voluntary tapping.

Clinical neurophysiological assessment can include: (i) prolonged measurement of frequency and amplitude of the tremor, generally with an accelerometer; (ii) looking for the co-activation sign; (iii) measurement of two body parts simultaneously; (iv) entrainment testing; (v) monitoring tremor while asking a patient to make a sudden rapid movement (ballistic movement) of another body part; and (vi) response to weighting the limb.

3.1. Frequency and amplitude

Accelerometry is best method to record frequency and amplitude, although EMG can also be used. Psychogenic tremors can have marked variation in frequency and amplitude measured over minutes.[26] Frequency is a better measure than amplitude since amplitude also varies moderately in organic tremors. There are no formal ranges for the reported variation and analysis is subjective.

3.2. Co-activation sign

In some patients with psychogenic tremor, the tremor is initiated on a background that begins with simultaneous tonic contraction of the antagonist muscles that will be involved in the tremor.[25] This increases the stiffness of the relevant joint, and it might be easier to have a tremor produced on this background. Since the resonant frequency of a joint increases with stiffness, and it is easier to drive a mechanical system at its resonant frequency, this would at least allow a faster frequency of tremor than might be possible otherwise.

3.3. Measurement in two body parts simultaneously

Organic tremors, although similar in frequency throughout the body, have slightly different frequencies in different body parts. The explanation for this is not clear, but the generators of both essential tremor and Parkinson tremor must be somehow fractionated. There are some

uncommon exceptions. Essential tremor rarely has the same frequency in the two arms, yet a rare disorder, orthostatic tremor, always has exactly the same frequency in different body parts.

Psychogenic tremor almost always has the same frequency in different body parts.[26] This includes simultaneous changes in frequency when they occur. A nice way of demonstrating this is with coherence analysis.[27] Coherence gives a quantitative measure of the similarity of phase of the two accelerometer signals.

3.4. Entrainment

Clinical neurophysiological measurements can objectify and quantify the amount of entrainment of the “involuntary” tremor by voluntary tapping of another body part. Most commonly this is done by measuring tremor of one hand and performing voluntary tapping with the other hand at a series of different frequencies. The different frequencies can be demonstrated for the subject with a metronome.

The first observation is the ease at which voluntary tapping can be done.[28] Patients with PMD have trouble matching the requested frequencies. The second, and more important, observation is what happens to the frequency of the tremor (Fig. 2), which might change, stop, or match the voluntary tapping completely, a clear outcome to interpret. It is important to judge the matching on the performance of the voluntary tapping, not on the metronome (since the voluntary tapping might not match the metronome). Coherence analysis is an excellent way to assess this similarity.

Parkinson tremor and essential tremor maintain their frequencies in this situation and do not entrain. By using clinical neurophysiologic studies, including entrainment, we showed that a patient with apparent essential palatal tremor had a PMD.[29]

All psychogenic tremors do not entrain. In some situations the psychogenic tremor remains at a stable frequency;[30] for instance, when the tremor is locked into a specific frequency by the mechanical conditions. An example is similar to the leg tremor, sometimes called voluntary clonus, which many normal persons have when they are anxious. When a person is sitting with the toe on the ground, a minimal effort of flexion/extension of the ankle will cause a fixed frequency of the leg bouncing up and down. Another possibility is that the tremor is so much “overtrained” by the central nervous system that it runs automatically and is not perturbed by another voluntary movement. While dual tasks ordinarily interfere with each other, automatic movements can escape this interference.[31]

3.5 Ballistic movement test

The ballistic movement test is similar to the entrainment test in that it assesses what happens to a trembling limb upon making a quick movement of another body part. The description of the test is that the tremor will transiently stop during the ballistic movement for a psychogenic tremor, but that this does not happen for either Parkinson tremor or essential tremor (Fig. 3). [32] Our own attempts to reproduce this test show that there are some false positives and negatives, so interpretation should be made with care.

Another variation in dual task interference is that reaction time would be slower for voluntary tremor or psychogenic tremor. This has been demonstrated, while no effect was seen in patients with Parkinson tremor or essential tremor.[33]

These studies can help make the diagnosis of psychogenic tremor, and the entrainment test is definitive when positive. The studies also give some insight into psychogenic tremor. Since psychogenic tremor has a similar frequency and phase in all limbs where it manifests, then there must be only a single generator. Additionally, since voluntary tapping (voluntary tremor)

can drive this generator, the generator must be the same as (or be under strong control by) the voluntary movement generator. This conclusion is similar to that from psychogenic myoclonus: psychogenic movement uses voluntary machinery to be produced.

4. Psychogenic dystonia

Much less is understood about psychogenic dystonia than psychogenic tremor. Clinically it is often difficult to decide whether a patient has psychogenic or organic dystonia. Authorities have also held different views over the years, and at one time most patients with dystonia were thought to have psychogenic dystonia. Dystonic movements usually look like voluntary movements, and task specificity, the phenomenon where the dystonia manifests only in specific tasks, often appears bizarre and “psychological”. Moreover, the physiology of dystonia itself is not fully understood.

There have been many studies seeking to understand the physiology of dystonia.[34-37] Most of these have enrolled patients with focal hand dystonia, although some have used patients with other focal dystonias and rarely patients with generalized dystonia. Physiologic abnormalities have been identified at the spinal, brainstem and cortical levels. A general summary is that inhibitory mechanisms are often faulty. Such mechanisms have been investigated with reflex studies and transcranial magnetic stimulation (TMS) techniques. Interesting, and curiously, most of these abnormalities are also present in asymptomatic body parts, not only where dystonia is manifest. The clearest situation is in focal hand dystonia, where the abnormalities are often found in the asymptomatic hand to the same extent as in the asymptomatic hand.

Clinical distinction between organic and psychogenic dystonia has few good clinical clues. The onset of psychogenic dystonia is often abrupt or it can be paroxysmal. One feature that often seems to indicate psychogenicity is that the dystonia is fixed, particularly from the start. Organic dystonia tends to be action induced, especially at the beginning.

There have been few investigations of psychogenic dystonia. Such studies are difficult: one reason is the difficulty of patient diagnosis. Another reason is that many physiological tests require the subject to be at rest. Patients with dystonia may not have relaxed muscles and this is particularly true of psychogenic dystonia, where the dystonia might well be fixed. A relevant physiological issue is that of co-contraction of antagonist muscles. While dystonic movement is usually characterized by co-contraction,[38] this is not always the case.[39] Hence, while lack of co-contraction might suggest psychogenicity, this is certainly not a definitive observation.

With these caveats in mind, one study has explored many abnormal inhibitory processes in organic dystonia. Surprisingly, patients with psychogenic dystonia had similar abnormalities.[40] The studies included short and long intracortical inhibition, cortical silent period, cutaneous silent period, and reciprocal inhibition in the forearm. How should these findings be interpreted? One possibility is that the data are tainted, but the investigators did their best to control the confounding problems. Espay et al. interpreted the data to indicate that the abnormal findings might result from the dystonia rather than being a cause. However, many of these abnormalities are found in asymptomatic body parts. Another possibility is that the abnormalities are the ones that predispose to dystonia, and that predisposed individuals can get organic dystonia or psychogenic dystonia depending on other contributing factors. In this regard, the question often asked is why a patient with conversion develops a particular movement disorder. This could be an answer in regard to psychogenic dystonia.

Another study has investigated patients with fixed dystonia.[41] The underlying etiology in these patients is often difficult to determine, but many do have psychogenic dystonia. Some

others have complex regional pain syndrome, and this is also obscure. In this study, patients with fixed dystonia were similar to patients with (typically mobile) focal dystonia. Short intracortical inhibition and the cortical silent period were similarly abnormal in the two groups. In this study, neither short nor long afferent inhibition was abnormal in either group.

There is a possible “light at the end of the tunnel”. A recent report showed that a measure of plasticity is abnormal in organic dystonia but normal in psychogenic dystonia.[42] Plasticity was measured with the paired-associative plasticity technique where frequent pairings of a median nerve shock and a TMS to the motor cortex at short intervals can lead to changes in motor cortex excitability. While plasticity was exaggerated in organic dystonia similar to prior studies,[43,44] it was normal in psychogenic dystonia. This certainly needs to be confirmed with another study.

5. Psychogenic parkinsonism

Psychogenic parkinsonism is uncommon and to our knowledge there have been no physiological studies. These patients primarily display extreme slowness. They may have tremor, and issues of tremor have been discussed earlier. Their slowness is often associated with a great sense of effort and rapid onset of severe fatigue. One way of helping to diagnose psychogenic parkinsonism is by 18F-fluoro-L-dihydroxyphenylalanine (fluoro-dopa) positron emission tomography scanning or dopamine transporter single-photon emission computed tomography scanning that should show normal dopamine metabolism in psychogenic patients. [45,46] One needs to be careful because such scanning will also be normal in guanosine triphosphate-cyclohydrolase-1 related dopa-responsive dystonia-parkinsonism.[47]

6. Psychogenic gait

Although psychogenic gait is a relatively common form of conversion, there are no physiological studies. Gait itself is complex and the disorders are highly varied, so it would be difficult to think of a physiological test that would be helpful other than to quantify the abnormal pattern of movement visible by eye. There are unusual patterns of stance and gait, often inconsistent, often dramatic, with lurching, but only rarely falls (and then without hurting themselves). Sudden knee buckling without falling is a common pattern.[48]

7. Genesis of PMDs and the meaning of “voluntary”

While the process of conversion remains largely a mystery, there are some data that emerge, particularly from psychogenic myoclonus and psychogenic tremor. The movement shares some mechanisms that produce voluntary movement. This includes the premotor cortex, supplementary motor area and motor cortex, structures that are involved in the most proximate movement planning and execution. The motor system does have difficulty doing more than one thing at a time. Tasks interfere with each other; interference of two tasks is dual task interference. Investigations show that voluntary movement and psychogenic movement interfere and interact with each other just like two voluntary movement plans. These motor structures are not the only factors in movement production: other parts of the brain need to motivate the movement first. It is this process that must differ in voluntary movement and psychogenic movement.

Voluntariness, what makes a movement voluntary, has been experimentally investigated recently.[49] From what we know about psychogenic movement, it is not the involvement *per se* of premotor and motor cortex in movement genesis that makes it voluntary. The interpretation of voluntariness seems to arise from a feed-forward neural signal, a corollary discharge. The origin of the feed-forward signal and where its message is received is not clear. There is the general sense that the feed-forward signal comes from frontal lobe structures,

perhaps including premotor and motor cortices. Processing this signal might be in the parietal lobe. Clearly some feed-forward signal needs to go to the parietal lobe so that anticipated movement can be compared to actual movement accomplished. One purpose is to refine movement accuracy. Another is to give rise to the sense of “agency” – that the person has produced the movement that has just occurred. Volition is the sense of willing a movement, and agency is the sense that the willed movement has occurred.

Recent observations from direct brain stimulation in patients during brain surgery provides general support of these ideas.[50] Stimulating the parietal area with low intensities produced a desire or an intention to move, while stimulating with high intensities could provoke the feeling that they actually had moved (when they had not). Stimulation of the premotor region could produce movement, but without any sense that movement had occurred. This is consistent with a feed-forward signal to the parietal cortex arising as movement is generated frontally. Then the sense of both volition and agency arises in the parietal area.

Neuroimaging studies might shed some light on the pathophysiology. There has been some neuroimaging of psychogenic paralysis. The findings, in mostly small series, have been confusing, but increased activation in frontal areas has often been seen. This has been interpreted, in a review of these studies, as an active inhibition of the motor system by the limbic system.[5] Evidence for abnormal limbic function is the enhanced startle in patients with PMDs with affective priming.[51]

Patients with psychogenic disease have no sense either of willing or agency (although if there is no willing, there cannot be a sense of agency). Hence it seems likely that in psychogenic movements, there is a failure of the feed-forward mechanism, but only for those movements that are psychogenic. The same patients can generate movements for which they have the sense of volition. Thus, the abnormality is likely in the source of the driving of the movement, proximal to premotor cortex, and the concomitant failure of a feed-forward signal. Data from our psychogenic tremor laboratory do show a decreased activation in parietal areas and decreased parietal connectivity with sensorimotor and limbic regions, consistent with these ideas.[52]

8. Conclusion

PMDs are an important clinical problem. Clinical neurophysiological testing can be helpful in diagnosing psychogenic myoclonus and tremor, and can lead to making the diagnosis definite. Further work is necessary in other types of PMDs. Additionally, PMDs are a valuable means of learning about voluntary movement itself.

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Fig. 1.

The movement-related cortical potential showing a normal-looking Bereitschaftspotential associated with psychogenic abdominal jerks. The trace is an average of 50 trials with zero time being the electromyography (EMG) onset of activity in rectus abdominus. Note the electroencephalography negativity at electrode Cz well before the EMG onset. In clinical practice the signal-to-noise ratio is often not as good as those commonly illustrated with hand movements because the movements may be unusual, like those here, and it may not be possible to get as many traces as desired. (Record obtained from an National Institutes of Health patient by Dr Codrin Lungu).

Fig. 2.

Measurements taken from accelerometric recordings from the right and left wrists showing entrainment with voluntary tapping. The results here are from a difficult case and show some of the subtleties of these observations. The patient's primary abnormality was a tremor of the right upper extremity. The left panels are recordings taken with both wrists extended. Tremor power is measured in $(\text{mg})^2$, which is one-thousandth of a gravity unit, squared. There is a tremor with peak frequency of 7.5 Hz in the right wrist. On the left side, there is a very low amplitude tremor at 6.7 Hz (note the scale of the power axis) that likely represents a physiological tremor. On the right-hand panels are the recordings of when the patient was asked to tap with the left hand at 3.0 Hz, matching a metronome. This is done well at 3.05 Hz and there is a prominent harmonic at 6.10 Hz. Looking at the raw accelerometric trace in the inset, the harmonic comes from a "notch" on the main peaks, apparently from a bounce as the patient was tapping on the table. The important observation is of the acceleration of the right wrist that shows a tremor frequency now at 6.1 Hz, with a sub-harmonic at 3.05 Hz. The change from 7.5 Hz to 6.1 Hz demonstrates the entrainment. The 6.1 Hz frequency, as seen in the raw traces, is a sinusoidal rhythm at twice the left-hand tapping rate. One tap is synchronous with the left hand, and the subsequent tap falls in the middle of the interval to the next tap, which is also synchronous with a left-hand tap. (Record obtained from a National Institutes of Health patient by Dr Dietrich Haubenberger).

Fig. 3. Multiple traces from accelerometer on the right wrist of the same patient as in Figure 2. The recordings show a brief pause in the psychogenic tremor with a ballistic movement of the other hand. The traces are aligned at 0 when there was the auditory cue for a ballistic movement of the left wrist, reaction time was about 200 ms, and the traces extend for 2 s before and after the cue. The pause in the right wrist tremor is clearly seen on every trace. (Record obtained from a National Institutes of Health patient by Dr Dietrich Haubenberger).