The laughter of the 1962 Tanganyika 'laughter epidemic'

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The laughter of the 1962 Tanganyika 'laughter epidemic'

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Abstract

The present article[1] *discusses the role of laughter in the much cited 'laughter epidemic' that occurred in Tanganyika in 1962. Despite its extraordinary nature, the veracity of the event is confirmed, crucially on the basis of similar reports. But most current representations are flawed by their exaggeration and misinterpretation of the role of laughter in the event, relating it to a humorous stimulus, a virus or environmental contaminant, or identifying it as contagious laughter. It is argued that the event is a motor-variant case of mass psychogenic illness of which laughter is one common symptom. Therefore it cannot serve as support for other arguments in humor research.*

Keywords: Laughter; laughter epidemic; mass hysteria; mass psychogenic illness; Tanganyika; Africa.

1. Introduction

In the literature on laughter, reference is often made to an instance of a 'laughter epidemic' that is reported from Tanganyika (now Tanzania), East Africa, in 1962 (e.g., Banwell 2000; Boss 1997; Brottman 2002; Cardoso 2003; Colligan et al. 1982; Conley 1963; Ebrahim 1968; Holden 1993; Kagwa 1964; Lambo 1965; Muhangi 1973; Provine 1992, 1996; Sirois 1982; Stearns 1972; Trump 2002; Wessely 1987). A recent citation that spawned much attention can be found in Provine (2000: 113ff). According to these accounts, the laughter epidemic originated in a girls'

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school, where students started to laugh uncontrollably, and subsequently spread to their communities to ultimately paralyze the whole country for several months.

Because this extraordinary instance, originally reported by Rankin and Philip (1963), is described and misinterpreted frequently and recently not only in popular literature, but also in scientific work, a reassessment of its general veracity and the reported circumstances — in the light of similar phenomena and their analyses — is warranted. This paper will argue that the event is much quoted for two reasons: On the one hand, it is a good story with an ironic juxtaposition of a joyful symptom and a disease event. On the other hand, it appears to provide support to several common-sensical notions about laughter, in particular its contagious nature, psychopathological correlates, and cultural universality.

The main misapprehension about the event arises from the central fact that the epidemic involved laughter: The assumption is that it must thus have been related to humor, on the one hand, and enjoyed, on the other. In fact, the laughter had nothing to do with merriment or humor nor was its contagiousness instrumental in the epidemic spread. Laughter, in this case, was just one and a fairly common symptom of an uncommonly large case of mass psychogenic illness (MPI), or mass sociogenic illness, that will have to be reassessed to highlight these most common misinterpretations of the 'laughter epidemic.' In general, despite the size of the event, both in the unusually large population affected and the long duration, most reports — except for the original one — are exaggerated, oversimplified and tendentiously ignore central facts and pertinent interpretations.

It has to be cautioned that for these reasons, mainly the non-humorous nature of the laughter involved, the 'laughter epidemic' itself may appear to fall outside of the purview of humor research. But this article, which aims to address this very point for the sake of humor research, is of course well within its limits. Accordingly, the present discussion is relevant mostly for humor scholars, but also psychologists and sociologists, whose fields centrally contribute to an understanding of the likely circumstances of the event, as well sociologists and anthropologists of science, who will find in the reception and distortion of the original report a fairly typical case of misquoting motivated by wishful thinking. In short, the good news is that there is a real event underlying the reports, but the bad news is that it had nothing to do with humor and only very little with laughter.

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2. Current reception

I choose to introduce the epidemic by way of its more inaccurate, but more recent citations. This reproduces the usual order in which the reader becomes acquainted with the episode and underlines the intended emphasis of the present discussion, which lies not only on the event itself, but also on its presentation and reception. The most detailed, carefully presented, and original source will be presented in depth in the following section.

The aim of this paper is not to discredit the journalists, like Trump (2002), who seems to have used the archive of his own paper that contains Conley (1963), or Sebastian (2003), who interviewed this author at a presentation of the present research. They largely depend on the work of the researchers and their purposes are those of journalists: to report the researchers' results and opinions, and to entertain. But since they are a main source, even for some academic approaches, their simplified accounts of the event dominate its reception. Banwell (2000) is a typical example:[2]

> It was 1962 in Tanganyika. A group of teenage girls were hanging out together. Something made them giggle — maybe someone told a joke, maybe they were giddy after a long day of school? The giggles quickly escalated from guffaws to belly laughs to shrieks to wild hysterics. The girls laughed, then cried, then laughed some more. When they met up with neighbors, they started to laugh too. The laughter spread from community to community. Soon, Tanganyika was caught in a full-scale laughter outbreak. Those people who did get to sleep woke up laughing again. Work halted; schools were shut down. The laughing in Tanganyika lasted for six months!

As I do not want to develop the present discussion against the background of an obvious strawman, let us turn to a more typical example: Holden (1993), referring to Rankin and Philip (1963), claims "[t]he authors wrote an account of a *delightful* social phenomenon in which whole African villages would be infected *en masse* by highly contagious bouts of laughter." While it would be desirable for an author of self-help literature (cf. Holden's webpage: http://www.happiness.co.uk/) to find evidence for such a "delightful" phenomenon, delight is far from the feeling of despair and confusion Rankin and Philip report the victims of the epidemic to have experienced, as we will see soon. Holden's misrepresentation continues (1993: 82; my emphasis):

> The laughter would occasionally reach such a point that work and schooling would have to be postponed until the *merriment* died down. On one famous occasion, one or two pupils at a Catholic girls' school began to *giggle*. The *giggling* gathered pace and soon the whole class was *merry*. Teachers were *tickled* by this spontaneous, *joyful* outburst and joined in. Soon the whole school was swimming in a tide of laughter. Word spread to the village, and when mothers came to collect their children they too became dizzy with laughter and chuckles of *delight*.

In these sources we always find an emphasis on positive emotions that are claimed to have accompanied the laugher symptom, e.g., "merriment," "joy," "delight." As we will see, these emotions were not underlying the symptoms reported of the event.

Typical for a more reliable, yet still centrally flawed, account in scientific literature is Provine (2000: 130–131) who presents an accurate summary of Rankin and Philip (1963), with additional interpretations of his own:

> The first symptoms appeared on January 30, when three girls started laughing. The symptoms of laughing, crying, and agitation quickly spread to 95 of the 159 students [. . .] Although temporarily debilitating, the laugh attacks produced no fatalities or permanent aftereffects, but teachers reported students being unable to attend to their lessons for several weeks after a laugh episode. [. . .]
>
> Before finally abating two and a half years later, in June 1964, this plague of laughter spread through villages 'like a prairie fire,' forcing the temporary closing of more than 14 schools and afflicting about 1,000 people in tribes bordering lake Victoria in Tanganyika and Uganda.

Provine focuses on the contagiousness of laughter, which he considers to be the main factor in the epidemic (cf. Provine 1992, 1996; also Stearns 1972: 40). As we will see, the event rather illustrates the contagiousness of hysteria, of which laughter may be a symptom, in a predisposed population. Provine (2000) attracted much journalistic attention and when it is reviewed, the Tanganyika episode is almost always among the quoted topics, as a tale too good not to be told, a tale about laughter having been too much of a good thing.

Finally, a typical attempt at an assessment of the event with a different focus can be found in Cardoso (2003; cf. also http://www.humourwise.co.uk/). It aims to find physical causes for the symptoms reported of the epidemic, again, centrally for the laughter involved:

> I find it improbable that a purely psychological mass reaction would last so long and be so widespread. [. . .] The American neurologists Hanna and Antonio

Damasio suggest that abnormal laughter occurs when structures in the basal part of the brain are damaged. The pathways that normally automatically adjust the execution of laughter to be appropriate to the stimulus for it are disrupted and the brain gets incomplete information about the cognitive and situational context of a potential stimulus — it gets it wrong about whether or not to laugh — resulting in chaotic behaviour. Based on this model, I suggest that a viral infection, probably some kind of encephalitis in the basal part of the brain, provoked the 1962 epidemic.

I will not pursue such physical cause explanations further. The details of the spreading pattern clearly exclude a viral infection as a potential explanation, and pathological laughter and crying show no contagiousness and a different progression (see 5.1).

Setting aside the information on details like location, affected population, and duration until their detailed report from the original source in the next section, the interpretation of the events in current sources presents the following picture, reflecting the most common notions about the event: Young females laugh, possibly because of a humorous stimulus, but they also cry, both with increasing intensity; these symptoms spread, possibly by contagion or a viral infection, interrupting everyday life and lasting between 6 and 30 months; despite their graveness and duration the symptoms are accompanied or even caused by merriment. The present paper aims to correct these notions and to suggest a different interpretation of the events on the basis of the original and other contemporary sources and more convincing analyses of the 'laughter epidemic' as a case of mass psychogenic illness.

3. Contemporary sources

3.1. *The original report*

Since it is the central source on which all other accounts rely and the first published description and careful assessment of the epidemic, I now turn to the concise[3] report by Rankin and Philip (1963) in detail in this separate subsection. Their reports begins as follows:

> The disease commenced on 30th January, 1962, at a mission-run girls' school at Kashasha village, 25 miles from Bukoba [...] when three pupils commenced to act in an abnormal manner. From that date until the 18th March, 1962, when the school was forced to close down, 95 of the 159 pupils had been affected.

> Fifty-seven pupils were involved from the 21st May, when the school was re-opened, until it was again shut at the end of June. (1963: 167)

The further progression of the epidemic can be divided into five stages. As the report is high in content of factual data, for the sake of clarity it is best presented in tabular form:

3.1.1. *Initial breakout*

Kashasha (25 m north of Bukoba) boarding school for girls (dormitories)
prodromal group: 3 students
symptoms: Attacks of laughing and crying lasting for a few hours, in a few cases up to a maximum of 16 days, with an average of 7 days, followed by a respite and then a recurrence in the majority of cases; general restlessness, persecution complex; no clear physical symptoms.

1. First phase
   beginning: 1/30/1962
   end: 3/18/1962
   duration: 48 days
   number: 95/159 pupils
   [school closed between phase 1 and 2]
2. Second phase
   beginnning: 5/21/1962
   end: ca. 6/31/1962
   duration: ca. 47 days
   number: 57/159 pupils

3.1.2. *Spread through cases from A. who were sent or went home*

1. Nshamba village (55 m west of Bukoba)
   beginnning: ca. 3/28/1962
   end: ca. 4/30/1962
   duration: ca. 34 days
   number: 217/10,000 villagers (school children, young adults of both sexes)
2. Ramashenye girls' middle school (outskirts of Bukoba)
   beginning: 6/10/1962
   end: 6/18/1962
   duration: 8 days
   number: 48/154 pupils

3. Kanyangereka village (20 m south of Bukoba)
   concurrent with B.2.
   number: 3 (family of a case from B.2. who was sent home)
4. Further spread at the time of the writing of report, 2 further school closures (boys' schools)
5. Milder occurrence in a Mbarara primary school (Uganda, 100 m north of Bukoba)

After the synopsis of the facts, Rankin and Philip turn to a preliminary analysis. It is still relevant today as their argument proceeds cautiously, refrains from speculation and, as I will argue, already points in the direction of a general explanatory model, which will be found most appropriate. According to the report, in order to exclude potential physical causes, selected subjects were tested for food poisoning and signs of toxic substances, with negative results. No known form of virus is assumed to account for the symptoms. Although I consider it unlikely, the possibility of a viral infection can, of course, never be excluded on clinical grounds. Yet, in view of the further argumentation of the present paper will make clear that it is an unlikely explanation.

Rankin and Philip conclude: "It is suggested that this is mass hysteria in a susceptible population. This is probably a culturally determined disease." (1963: 170). Accordingly, the next section will briefly present related and similar cases of culturally determined diseases, that is, MPI, previously known as mass hysteria, before I will continue to outline the relevant research on MPI in general and to analyze to what degree it pertains to the case at hand, including a focus on the cultural determinants of the Tanganyika 'laughter epidemic.'

3.2. *Similar cases*

This subsection presents similar contemporary cases of MPI in the vicinity of the event under discussion here. This will serve two purposes: First, as crucial support for its general veracity, it will establish that the Bukoba case and its spread is not a singular and isolated event. Second, it will show that laughter is just one of several symptoms in these additional cases, as much as in the Bukoba event itself, all of which are common for a specific variant of MPI.

Kagwa (1964), citing Rankin and Philip (1963), and Ebrahim (1968) discuss the Bukoba case as an instance of three connected events, the

other two being "running manias," which broke out in the area around Lake Victoria. One outbreak occurred in Kigezi (southwestern Uganda) in July 1963 with ca. 600 affected people. The second instance took place in Mbale (eastern Uganda) in November 1963 involving about 300 victims. Both cases are characterized by aimless running, general hyperactivity, as well as violence, and these symptoms spread predominantly among school populations in a pattern identical to the Bukoba event. A further similar event is reported by Muhangi (1973) for Ankola (southwest Uganda) in July 1971. Fifty of the 287 students of a Rugarama male primary school show grimacing, vulgar language, and aimless walking, as well as laughing. Another similar, but much shorter, incident from neighboring Zambia is reported by Dhadphale and Shaikh (1983).

With the help of these reports, it can be established that related events took place in the same region in the same period and that additional similar events are reported. In addition to placing Rankin and Philip's report into context, these reports point to the main problem with the alleged laughter epidemic, namely that it didn't just involve laughter, but "[t]he epidemic was characterised by episodes of laughing *and crying* (Rankin and Philip 1963: 167; my emphasis). That is, first of all, it is not a *laugh* epidemic, but an event involving seeming expressions of strong emotional disturbance in general and, in some specific cases, laughter in particular. As such the laughter found in this event is clearly not elicited by humor, but rather a symptom of mass psychogenic illness.

4. Mass psychogenic illness

4.1. *General*

This section will establish the complex of mass psychogenic illness (MPI), its general features, populations typically affected, patterns of spreading from initial to later stages, general variants, and possible causes. All these aspects are closely related so that the argument will repeatedly have to anticipate certain points in later subsections. The focus in the discussion will be on factors that pertain to the initial outbreak (A. in section 3.1). Seminal surveys of cases have been compiled and analyzed by Sirois (1974; reported in Sirois 1982), and a follow-up by Boss (1997), as well as the work of Bartholomew and associates (e.g., Bartholomew and Wessely 2002) and Wessely (e.g., 1987). In addition to numerous case

studies reporting events similar to our focal case, these surveys are the main sources for this section. It will become obvious that MPI is a paradigm example for Fleck's (1981 [1935]) observation that diseases are constructed models on the basis of pseudo-theories of causes and symptoms; that is, they are functionalist answers to urgent and often biased questions. Since the Bukoba case is an almost prototypical example of MPI, and references abound in the work introduced in this subsection, it is assumed necessary to introduce another additional detailed example in this section.

The following list summarizes the characteristics of the typical course of MPI (Seldon 1989: 893):

- absence of physical findings about organic causes
- preponderance in girls/women and (pre-)adolescents
- transmission by sight/sound
- hyperventilation/syncope (as signs of anxiety)
- rapid spread and remission of symptoms
- relapses in original settings of outbreak

as well as the generally assumed underlying causes:

- unusual physical/psychological stress in general
- evidence of prior physical or mental stress of the prodromal cases in particular

Based on these general observations, I will discuss, in turn, those characteristics in more detail that are relevant for the Bukoba case.

4.1.1. *Affected population.* All surveys over case studies confirm that "the prevalence of illness is almost always higher in females than in males." (Boss 1997: 235). Sirois (1982), for example, observes that "[o]f the 70 reviewed outbreaks [in Sirois 1974] 34 appeared in schools, [and] [w]omen were almost exclusively involved (80%), [. . .] [t]hey were young, below 20 years of age, often in the first years of adolescence" (104). The reason for the prevalence of females in affected populations is not completely obvious, but the general disenfranchisement of females in most cultures leading to higher psychosocial stress is assumed. McGrath's argument is typical:

> Perhaps the high incidence of females, as both affected and non-affected members of the settings in which MPI occur, simply reflects our tendency to put females in low status and otherwise unrewarding jobs. If so, then 'female,' along with

> 'young' and 'poor,' should be treated as a variable that signals 'low social status,' not some character flaw like 'hysteria' or 'hypochondria.' Perhaps the MPI symptoms represent a 'last resort' means for low-status persons (female, young, poor, etc.), in high stress situations, to express their distress. (1982: 73)

The gender question is not central to the present study, but it can be observed that the unequal gender and age distribution is reproduced in the Bukoba case. I will return to the more general point that MPI is a last resort to escape stress situations for powerless "low status" people below, as it is a key part of the present argumentation.

4.1.2. *Settings.* It is not astonishing that school settings are often indicated for MPI, as a population of young age is likely to congregate in such a setting. But there are specific additional factors that make a school population a likely breeding ground for MPI, independent of gender distributions and partially independent of age. Sirois observes that "some outbreaks in the school setting occurring at the beginning of the school year could be seen as 'rites of passage.' They would be useful to bind anxiety linked to the formation of the group and act as witness of collective cohesiveness" (1982: 106). Furthermore, in the case of boarding schools, where students are living together around the clock, stress is intensified through the separation from family settings. An additional factor is observed by Boss: "Outbreaks in schools may have been reported more frequently than those that occur elsewhere because of the importance attached to investigating outbreaks involving children" (1997: 239), an argument also valid for the many reported MPI cases in Western schools (e.g., Helvie 1968; Jones et al. 2000; Small and Borus 1983). Yet another reason may lie in the fact that in colonial settings, as in the Tanganyika case, schools are the point of contact between the traditional local population and Western teachers/missionaries. As we will see below (subsection 4.2) such a point of contact is also a point of friction, leading to additional stress. Furthermore, the local population may in general be susceptible to MPI, and may just be the fact that a Western observer happens to be present to document the outbreak.

4.1.3. *Spread.* In the initial or prodromal stage, before the epidemic spread, the first or *index* cases involved are often "unusual ['hysterical'] personalities," easily affected by stress; this facilitates spread to 'normal' personalities (Kerckhoff and Back 1968: 40). In contrast to this, Sirois

sees the typical pattern involving hysterics rather as second and third cases after the index case (1982: 112), while the index case is not necessarily susceptible to hysterical reactions, but more importantly of high social status. Teoh and Yeoh (1973: 288) confirm that in the prodromal stage, the index case is usually dominant in the given population, and the second and third cases are troubled personalities in the orbit of the index case (cf. also Benaim et al. 1973). This sociometric pattern, in which "the regressive effect of crowd behavior on intellectual and emotional processes — and a small minority of unstable or emotionally labile individuals which often can be found to gravitate around index cases" (Sirois 1982: 104) — plays an important role, can unfortunately not be confirmed for the Bukoba event. The the relevant data has simply not been recorded by Rankin and Philip, although "three pupils" were identified as the initial group affected (1963: 167).

The general spreading pattern of MPI after the prodromal stage is characterized by a snowballing effect with early satiation, the quick drop-off being accelerated through outside forces (Kerckhoff and Back 1968: 35). At first sight, this appears not to be the case for the Bukoba event, which lasted long without drop-offs, yet these are better understood as relapses, in particular the case in the original population (A.2 in subsection 3.1) and in individuals. Rather, Sirois identifies the Bukoba epidemic as a case of "diffuse outbreak" (1982: 107) combining the spread pattern of closed and open settings: "Its prodromal stage is obscure, but the epidemic spread starts in the typical explosive manner in a closed setting, the school, but then spreads into the community, and the subsequent "rebound stage is spectacular" (ibid.).[4] The Bukoba event started in the school environment and then spread into the communities/families (cases under B. in subsection 3.1), where it lingered. Thus, it appears to combine the propelling factors of the two typical developments: "the most common outbreaks — those in schools and places of business — tended to be of short duration, whereas those in communities and families tended to last longer" (Boss 1997: 238).

The general problem of the lack of information on the social composition of the affected population in the Bukoba case is grave and obvious, as Sirois recognizes: "When the outbreak lasts longer (Kagwa 1964; Rankin and Philip 1963), it is often found that the reservoir of susceptible persons is much larger than originally suspected. this meant that important and more general aspects necessary for the understanding of the situation were missed and the original group was only instrumental in the

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outbreak'' (1982: 108). This points to the importance of identifying not only clinical aspects and the underlying triggering event of the epidemic, if any, but also the sociocultural context, which Rankin and Philip (1963) were not able to do sufficiently. This is not so much a fault of oversight, but the Bukoba case occurred when the model of MPI had not yet matured as a concept, and a main issue in 'mass hysteria' research was to eliminate potential physical causes.

4.1.4. *Anxiety vs. motor variant.* In the previous paragraphs, different types of MPI were described mainly with respect to their development. But a more important variance is characterized by different types of symptoms. This is reflected in Wessely's (1987) convincing distinction of two main types of MPI: ''mass anxiety hysteria covers outbreaks demarcated by the phenomena of anxiety abdominal pain, chest tightness, dizziness, fainting, headache, hyperventilation, nausea and palpitation'' (1987: 112) and ''mass motor hysteria,'' which such symptoms as seizures, drop attacks, hysterical dancing, running, and, in three cases, described in Dhadphale and Shaikh (1983), Ebrahim (1968), and Rankin and Philip (1963), respectively, also laughing. Accordingly, Wessely classifies the Bukoba outbreak as ''mass motor hysteria'' (1987: 112). A difference corroborating this classification is that, in contrast to anxiety types, motor hysteria attacks ''may persist for months or even years'' (Wessely 1987: 113), which holds for the case under scrutiny here and makes the account of the episode appear less fantastic. A case described in (Nandi et al. 1985) is assumed to have involved relapses over a period of ten years.

The distinctive symptoms of motor-variant MPI must be discussed in connection with the underlying causes, namely the possibility of escape from stress situations, which will be attempted below (subsections 4.2). The motor variant can be used for that purpose only if its more extreme symptoms are accepted as symptoms of illness in the cultural context in which they occur. This social acceptability of motor-variant symptoms appears to be lower in Western cultures. These cultures have 'clinicalized' scripts of disease, into which symptoms like dancing, laughing and running cannot be accommodated. This would also explain, why in the motor-variant epidemic originating in Bukoba ''[n]o literate and relatively sophisticated members of society have been attacked'' (Rankin and Philip 1963: 167). From a historical perspective, Boss points out that in the period from 1872 to 1972, surveyed in Sirois (1974), motor-variant

symptoms are more commonly reported, while in the period from 1973 to 1993 anxiety variant symptoms prevail (1997: 238). In short, today and in so-called Western societies, dizziness and fainting can earn you a day home from work, while laughing and dancing will earn you odd looks. In the period until 1972 and in so-called traditional societies, laughing and dancing will also have been considered as symptoms of disease.

4.2. *Causes: Sociocultural transition and stress*

Rankin and Philip stress that "[t]he type of mental disorder that affects a community is influenced by the culture of the particular community" (1963: 170). More specifically, as we saw in the previous subsection, the culture of the community also strongly predicts the variant of MPI that may occur in it. These observations about types of symptoms point in the direction of a meaningful explanation for underlying causes of MPI, which will turn out to be more complex and tentative than previous attempts in humor-related and other literature that have aimed to reduce it to contagion or viral infections. Sirois summarizes that a "state of ideological or cultural transition is frequently noted to be associated, as well as periods of uncertainty and social stress like wars, endemic diseases, or technological changes" (1982: 106). Tanganyika clearly underwent such a period at the time of the epidemic. Therefore, the analysis of the Bukoba event in terms of cultural factors and stressors will be facilitated by a brief outline of the political situation in Tanganyika (cf. Ofcansky and Yeager 1997), before I will turn to the specific local factors at the mission-run boarding school in Bukoba and similar settings.

The area of Tanganyika was a colony since the late 1880s. It was controlled first by Germany, officially as "Deutsch-Ostafrika" since 1891, and after World War I became a mandate territory of Great Britain in 1919 and practically part of the British Empire. Like in other African colonies, no strong independence movement developed until after World War II, when the U.N. as the successor to the League of Nations renewed the mandate to Britain with the obligation to prepare the country for independence. The most prominent African figure in the intensifying struggle for Tanganyikan independence, Julius K. Nyerere, formed the openly anticolonial Tanganyika African National Union (TANU) with collaborators in 1954. During Nyerere's term as first Prime Minister, the country achieved full independence on December 9, 1961. It appears

likely that these political events, less than two months from the outbreak of the Bukoba event on January 30, 1962, and in addition the abandonment of racial division in schools since January 1, 1962, increased the sociocultural stress situation in the young country in general (cf. Wessely 1987: 114). Very similar developments occurred to the neighboring countries, mainly Uganda, independent since 1962, but also Rwanda, Burundi, Kenya, and Zambia, from which the related motor-variant epidemics are reported (cf. subsection 3.2).

As briefly mentioned above, the local situation in the school setting can also increase stress as it is a point of friction and transition where the students from the traditional tribal society are confronted with Western methods of instruction, educational expectations, and religious-moral values (Boss 1997: 234). In addition, the transition of the students through adolescence takes place while they are separated from their families. Stearns agrees that these factors result in "anxiety, guilt feeling, loss of identity, feelings whose repression by schooling and discipline finally resulted in aggressive-compulsive behavior or conversion hysteria" (1972: 43), his term for a specific type of MPI.

The high indication for MPI of the specific situation at boarding schools in countries in transition is corroborated by the research on motor-variant cases by Teoh and colleagues (e.g., Teoh and Yeoh 1973; Teoh et al. 1975). Teoh and Yeoh (1973) focuses on cultural transition as the central stress factor for a group of young college women involved in a MPI episode in Malaysia. In particular the way these changes affect the educational system is considered a crucial environmental factor for MPI (1973: 284):

> [W]ith higher educational expectations of the Malay rural parents for their children, greater pressure is imposed on these children [...] These adolescent girls, away from home, seek alternative culturally-sanctioned modes of expressing their frustration in this transitional period, in the form of outbreaks of epidemic hysteria.

Sirois summarizes the general factors of this endemic propensity, which correspond to observations of Muluka et al. (1985: 251) for a similar Kenyan case, as follows: "outbreaks in Malaysia were detected in schools in 1970–1971, after the 1969 troubles and in a context of administrative and educational changes" (1982: 109).

In sum, we find a transitional stress-inducing situation both in the country, which is in the process of consolidating its recently gained

independence, in general and in the specific circumstances of the affected population in particular, namely, separation from family, adolescence, and confrontation with Western educational and other standards. Which of these factors is more important, and which may not even pertain, and which may have been overlooked, especially at the individual and prodromal group level, can no longer be clearly decided for the Bukoba event. But I argue that a combination of these factors creates a stress-inducing context that is the indirect cause of the epidemic event described by Rankin and Philip (1963).

4.3. *Potential purpose: Advantages of the sick-role*

Although it is neither instrumental for the assessment of the veracity of the reports on the Bukoba case, nor for the analysis of the role of laughter in the event, a central point for the general understanding of the epidemic as a case of MPI is the question for possible motives of the affected population. Since they are experiencing a disease event, the victims are normally not consciously aware of the reason for their symptoms: "The common feature of the stressors underlying outbreaks of mass motor hysteria is an inability on the part of the subjects either to comprehend the true nature of the threat facing them or to avoid it" (Wessely 1987: 115). This subsection will accordingly be the most speculative and controversial[5] of the present study.

As briefly mentioned above and in accordance with most research on MPI (e.g., Boss 1997; Kerckhoff and Back 1968; Wessely 1987), I see the reason for people suffering from severe stress and anxiety to exhibit the symptoms of MPI in that they may afford them an escape from the situation that induces the stress and anxiety. "Outbreaks provide a temporary escape from stress because factories, offices, or schools close while investigations are under way" (Boss 1997: 237). This advantage of MPI is identical to that of the sick-role in general: The sufferer can evade a situation that they are otherwise expected to endure. The central case of motor-variant MPI reported by Nandi et al. (1985) is remarkable in this respect: The eight women affected by the symptoms relapsed over more than a decade (1985: 248), and the instrumental nature of their episodes in escaping the beating by their alcoholic husbands is a shared stressor rather directly addressed by their behavior.

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The symptoms must be socially acceptable in a given cultural context for MPI to fulfill this function, which explains the difference of MPI variants presented above in two ways. First, as Kerckhoff and Back observe: "What occurs in cases of hysterical contagion is that physiological symptoms, which occur largely as a result of unresolved psychological stress, are explained (and thus responded to) in terms of a newly invented label" (1968: 34). In other words, the purported trigger — in Western societies for example, nuclear fallout, viruses, or environmental contaminants, in traditional societies for example, witchcraft and curses, poisoned food, or, insect bites — as well as the symptoms — anxiety symptoms and extreme motor behavior, respectively — that provide a socially acceptable rationalization of the socially unacceptable behavior of leaving the workplace, school, or any other stress-inducing situation, must be socially acceptable.

A very telling 'confession' by an index case of an epidemic was characterized by a falling symptom among adolescent students of a London school supports this escapism argument: "I enjoyed the attention this malady afforded and the general concern of everyone around me. [...] I used it as an escape from the problems I could not face at home and at school, and became completely wrapped up in it" (Benaim et al. 1973: 369). In sum, there are "rewards to be gained from being sick" (Muluka et al. 1985: 251). But these rewards can be reaped only if the sickness is presented as evoked by a socially acceptable cause and expressed in socially acceptable symptoms. Under these circumstances MPI can afford its victims the advantages of the sick-role. It must be cautioned, again, that the rationalization presented in this section is the most speculative part of the present discussion, and that the secondary sick-role advantage is usually not an explanation for all individuals involved.

5. Potential additional explanations

On the basis of the previous sections, it should have become obvious that the laughter involved in the Tanganyikan epidemic was one of the symptoms of motor-variant MPI. But before I can turn to a summary of the argument that leads to this conclusion, I want to briefly address additional research and explanatory attempts besides MPI that can help clarify what may have taken place in 1962 and what may not, both because the explanations pertain to the case and because they are thought to pertain to it, but don't.

5.1. *Pathological laughter*

A different and obviously oversimplified explanation is to declare the laughter involved in the Bukoba event to be pathological laughter of a specific type reported in other cases (cf. Black 1982). This kind of laughter is inappropriate, unmotivated, as well as involuntary (cf. Shaibani et al. 1994: 243) and continuous (Arlazaroff et al. 1998: 184). Apart from brain lesions (cf. Parvizi et al. 2001) and other diseases with organically distinct causes, psychiatric disorders that include pathological laughter and crying (PLC) as a symptom include hysteria. Arlazaroff et al. (1998) describe a patient who has fits of pathological laughter after hitting her head in a car accident. While no social transmission is involved, the spells of this patient occurred mainly in stressful or delicate situations [...]." (1998: 186). Thus, while PLC is a related symptom, it cannot account for the epidemic dimensions of the Bukoba event.

5.2. *Physiology of laughter*

The extent to which the laughter symptom is reported to have lasted during the epidemic event can also be evaluated from a physiological perspective, a research topic since Spencer (1860). Physiological aspects of laughter — prominently respiration and phonation — vary across genders, individuals, and also in the output of one individual along various factors (cf. Black 1984; Fry and Rader 1977; Hauser et al. 1997). In general it is an extremely exhausting activity, resulting in a signal that is more comparable in volume to shouting at up to 80 dB (Rothgänger et al. 1998) than to normal speech at ca. 60 dB. The muscles involved in the exhalation crucial for laughter — diaphragm, abdominal and rib cage muscles — are usually not active in that part of the breathing cycle (Ruch and Ekman 2001: 432) and thus not well trained for it. In addition, during laughter there is a pronounced antagonism between the opposing sets of muscles for inspiration and expiration, which are normally working alternately. This produces the extraordinarily high levels of expiration and subglottal air pressure in laughter, up to five times as much as in normal phonation (Ohala 1990; Schroetter 1925), which are usually followed by long periods of apnea (Lloyd 1938). Because of all these factors, humans tire quickly from the saccadic contractions required for laughter, as witnessed by the soreness of abdominal and thoracic muscles after extensive laughing and, vice versa, the painfulness of laughter after exercising that has

involved abdominal muscles. Laughing continuously for long stretches of time — as they are understood by some to be implied by the overall length of the epidemic event in Tanganyika — must be considered impossible. These gelotological results indicate that only short episodes of laughter can be symptoms of a the motor-variant of MPI. They could last for seconds at a time, and only be repeated over short stretches for each individual, but not over hours, weeks, or even months as claimed in some reports on the 'laughing epidemic.'

5.3. *Contagious laughter*

A seemingly plausible interpretation of the epidemic reduces the cause to the contagiousness of laughter (cf. Black 1982). This argument is represented most prominently by Provine (1992, 1996), and has received much attention since the publication of Provine (2000) and the subsequent resurgence of reports on the event: "The power of contagious laughter as a social coupling process is suggested by a persistent epidemic of laughter that began among 12- to 18-year-old girls in a boarding school in Tanganyika and spread throughout a district, requiring the closing of schools" (1992: 1). As we have seen in the preceding discussion, the event cannot be reduced to laughter as the central symptom. Thus, the undeniably contagiousness of laughter at short range, unsuccessfully modeled by Provine's laughter detector-generator (2000: 149), cannot serve an explanation for an event of the extent of the motor-variant MPI case that took place in Bukoba.

5.4. *Viral infection and environmental contaminants*

As we have seen above, there are attempts to explain the 'laughter epidemic' as a reaction to a virus (Cardoso 2003) or environmental contaminant. Rankin and Philip (1963) excluded a limited number of such possible causes. Since no blood samples are known to have been preserved, no update of their falsification of such causes can be attempted. Yet, I would argue that this is not necessary, as there are no known viruses or contaminants leading to the symptoms described for the event, while MPI provides us with a plausible theory.

In oral communication, similarities in symptoms — and more tentatively in cause — to Kuru have been pointed out to me (cf. also Conley

1963). Kuru, also called the "laughing sickness," is a well-documented (e.g., Gajdusek 1963, 1976) type of transmissible spongiform encephalopathy, in which prions transmitted through the ritual eating of the brains of recently deceased humans cause fatal brain deterioration. One common symptom in the final stages of Kuru are short episodes of laughter (cf. also Virani and Jain 2001). Yet, there is only one connection between the "laughter epidemic" and this "laughter disease": The ironic association of a sad event, a disease, with a seemingly happy symptom, laughter, in both cases leads to their analogous names.[6] This also reflected in phrases like "plague of laughter" (Provine 2000: 1313) and taken to the extreme in the title of Zigas (1990): "Laughing Death."

6. Conclusion

The Tanganyika laughter epidemic is a case of motor-variant mass psychogenic illness. This is the result of the present reassessment of the original report on the event and corroborated by reports on similar events under similar circumstances. Although emphasized in reports on the epidemic, laughter in this context is just one of several symptoms, even if it makes a descriptive name based on the ironic contrast between a sign of joy and merriment, on the one hand, and a painful disease event, on the other. Laugher played a much smaller role in the event than most current descriptions claim, not least because laughing on an epidemic scale is physiologically impossible. The laughter in the event is not caused by a humorous stimulus that transmits it. It is not a case of contagious laughter, neither as emotional or behavioral contagion, but only one of several common symptoms of motor-variant MPI, none of which could be triggered by or trigger laughter, e.g., running or seizures. For these reasons it is suggested that the present article should be the last to discuss the 'laughter epidemic' in research on humor, other than to illustrate the dissociation of laughter and humor.

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Notes

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1. The author would like to thank Robert E. Bartholomew, Willibald Ruch, and the two anonymous referees for helpful comments on earlier versions of this paper.

2. An even wilder account can be found in Shibles (n.d.): "Between 1962 and 1964 it is reported that 1000 people in Tanganyika and Uganda (especially girls in Catholic convent schools) died in a group laughing epidemic. The report, it would seem, would need detailed checking." Indeed.
3. It comprises only four pages, one of which is taken up by a map of the area around Lake Victoria.
4. Sirois (1982: 110) ascribes a report on an episode of "collective running [that] shows an underlying fantasy of being savagely attacked and contaminated by some atomic power." to "Rankin and Philip (1964) [sic!]." It is unclear which source he is referring to as no such explanations are proposed by Rankin and Philip (1963) nor by Rawnsley and Loudon (1964), the next entry in his bibliography that also corresponds in page number to his citation.
5. Ascribing psychological motivations is always a speculative business as well as one seldom well received by those whose role as sufferers from a disease it evaluates. A prime example for this is the controversy over the Joint Royal College Report on chronic fatigue syndrome, one of the authors of which is Wessely (Royal College of Physicians et al. 1996).
6. There is one further coincidental connection at the perimeter of Kuru that has no impact on the present argument: "Five young females, they had allegedly been typical advanced Kuru cases whose progressive symptoms had ceased. All five, like Teirari, were in close contact with other Kuru victims, and being of emotional, somewhat hysterical temperament, had developed what was obviously a hysterical mimicry of Kuru" (Zigas 1990: 285).

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