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Review

Genuine and drug-induced synesthesia: A comparison

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ABSTRACT

Despite some principal similarities, there is no systematic comparison between the different types of synesthesia (genuine, acquired and drug-induced). This comprehensive review compares the three principal types of synesthesia and focuses on their phenomenological features and their relation to different etiological models. Implications of this comparison for the validity of the different etiological models are discussed.

Comparison of the three forms of synesthesia show many more differences than similarities. This is in contrast to their representation in the literature, where they are discussed in many respects as being virtually similar. Noteworthy is the much broader spectrum and intensity with the typical drug-induced synesthesias compared to genuine and acquired synesthesias. A major implication of the phenomenological comparison in regard to the etiological models is that genuine and acquired synesthesias point to morphological substrates, while drug-induced synesthesia appears to be based on functional changes of brain activity.

Contents

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1. Introduction

Synesthesia (Greek: syn = together; aesthesis = perception) is usually defined as a crossing of sensory perceptions, where stimulation within one sensory modality/stream leads to an internally generated perceptual experience of another sensory modality/stream. The stimulated percept is called the inducer whereas the additional perceived percept is called the concurrent (Grossenbacher & Lovelace, 2001), and the type of synesthesia is named the inducer-concurrent pair (e.g. auditory–visual synesthesia where acoustic stimulation leads to a visual experience). Synesthesia is not restricted to inter-modal couplings but can also occur within a modality. An example is grapheme-color synesthesia where coupling between written letters and color is experienced.

According to Grossenbacher and Lovelace (2001), we can differentiate three forms of synesthesia:

1. Constitutional or genuine synesthesia.
2. Acquired synesthesia.
3. Drug-induced synesthesia.

In genuine synesthesia, the inducer-concurrent coupling is experienced the entire life.

Acquired synesthesia can be experienced after brain damage (Jacobs, Karpik, & Bozian, 1981; Ro et al., 2007) or sensory deafferentation (Armel & Ramachandran, 1999). In this type the concurrents are usually referred to as phosphenes. A phosphene (an inadequate stimulus to the photoreceptors (Kandel, Schwartz, & Jessell, 2000)), is a visual phenomenon elicited by stimulating the retina mechanically, electrically (Penfield & Rasmussen, 1957), magnetically (Eichmeier & Höfer, 1974), or by direct stimulation of the occipital cortex (Cowey & Walsh, 2000).

Drug induced synesthesia is experienced temporarily during acute effects of a hallucinogen (mescaline, psilocybin, LSD) drug intoxication (Beringer, 1927; Friedrichs, 2009; Shanon, 2002). During intoxication, a dream-like state of consciousness is typical, accompanied by changes in the relationship between the sense of self and the cosmic, an intensification of affectivity (Masters & Houston, 1966), a decrease of self-control, and a change in time perception and thinking abilities (Vollenweider, 2001; Wittmann et al., 2007). Additionally, an intense inner flow of sensations is often experienced accompanied with hallucinatory activity, especially in the visual sphere (Hoffer & Osmond, 1967; Studerus, Gamma, & Vollenweider, 2010). In reviewing all the manifold effects of hallucinogenic drugs, it becomes clear that synesthesia is only one possible aspect of the intoxication. A major effect of hallucinogenic drugs is the intensification of sensory perception, including illusions, pseudo-hallucinations, and, in very rare cases, true hallucinations (Leuner, 1962).

During the acute effects (cf. Table 1), the first phase of the intoxication usually induces hallucinatory phenomena that are more simple in design like abstract geometric forms (“entoptic phenomena” or “Form constants” (Klüver, 1966)). As the course of intoxication progresses (or with higher dosage), the visual phenomena change to more complex forms and may even develop into coherent scenes seen on a kind of “inner screen” (Friedrichs, 2009).

Much has been written about genuine synesthesia (Bleuler & Lehmann, 1881; Cytowic, 2002; Harrison & Baron-Cohen, 1995; Hochel & Milan, 2008; Hubbard, 2007; Marks, 1975; Mattingley, 2009; Ward & Mattingley, 2006), but the other forms have been somewhat neglected. Drug induced synesthesia is reported, but, until now, there were no systematic studies (see (Shanon, 2002)). The same is true for acquired forms. Most papers available are neurological descriptions of single cases where the type of damage and effect is reported (Armel & Ramachandran, 1999; Jacobs et al., 1981; Kim, Dryja, Lessell, & Gragoudas, 2006; Koike & Yoshino, 1990; Lessell & Cohen, 1979; Page, Bolger, & Sanders, 1982; Rao, Nobre, Alexander, & Cowey, 2007; Ro et al., 2007; Steven & Blakemore, 2004; Vike, Jabbari, & Maitland, 1984), but systematic investigations are missing.

All these phenomena are discussed separately and are not compared directly. Yet such comparisons are of importance in order to shed light on the mechanisms underlying synesthetic perception. Is the phenomenology of the different types comparable? Are there commonalities in the current etiological models? Can knowledge about one form be transferred to the others? Or are these types too different to even speak of a “unitary” phenomenon?

Table 1
Visual and synesthetic phenomena during the course of acute hallucinogen effects (psilocybin 20 mg p.o.) (Grof, 1975; Heimann, 1961; Leuner, 1962).

	30–75 Min	>75-Min	240–350 Min.
Visual phenomena	Abstract geometric patterns Klüver's form constants entoptic phenomena	Complex organic imagery Scenic imagery	Decrease and fading of imagery
Synesthetic phenomena	More automaticity	Less automaticity	