

Mexican waves in an excitable medium

The stimulation of this concerted motion among expectant spectators is explained.

The Mexican wave, or *La Ola*, which rose to fame during the 1986 World Cup in Mexico, surges through the rows of spectators in a stadium as those in one section leap to their feet with their arms up, and then sit down again as the next section rises to repeat the motion. To interpret and quantify this collective human behaviour, we have used a variant of models that were originally developed to describe excitable media such as cardiac tissue. Modelling the reaction of the crowd to attempts to trigger the wave reveals how this phenomenon is stimulated, and may prove useful in controlling events that involve groups of excited people.

Using video recordings, we analysed 14 waves in football stadia holding over 50,000 people. The wave (Fig. 1) usually rolls in a clockwise direction and typically moves at a speed of about 12 metres (or 20 seats) per second and has a width of about 6–12 m (corresponding to an average width of 15 seats). It is generated by no more than a few dozen people standing up simultaneously, and subsequently expands through the entire crowd as it acquires a stable, near-linear shape. (For details and interactive simulations, see <http://angel.elte.hu/wave/>.)

Because of the relative simplicity of the Mexican wave, we were able to develop a quantitative treatment of this type of collective behaviour by building and simulating models that accurately reproduce and predict its details. We found that well-established approaches to the theoretical interpretation of excitable media^{1–3}, which



Figure 1 The Mexican wave, or *La Ola*, sweeping through a crowd of spectators. A few dozen fans leap up with their arms raised and then sit down as people in the next section jump to their feet to repeat and propagate the motion.

were originally created to describe processes such as forest fires or wave propagation in heart tissue, can be generalized to include human social behaviour.

We developed two mathematical simulation models, one minimal and one more detailed. By analogy with models of excitable media, people are regarded as excitable units — they can be activated by an external stimulus (a distance- and direction-weighted concentration of nearby active people exceeding a threshold value, c). Once activated, each unit follows the same

set of internal rules to pass through the active (standing and waving) and refractory (passive) phases before returning to its original resting (excitable) state.

The simpler model distinguishes only three states (excitable, active and passive) and accounts for variations in individual behaviour by considering the transition probabilities between these states. The more detailed model takes into account an actual deterministic activity pattern. The two versions differ in the way in which stochasticity (that is, differences and fluctuations) in the above behavioural patterns is represented (for details, see <http://angel.elte.hu/wave/>).

We applied these models to investigate the conditions that are necessary for triggering a wave. Figure 2a shows the evolution of a wave provoked by the simultaneous excitation (rising to their feet) of a small group of people. By using parameters deduced from video recordings for the different sizes and characteristic times of the phenomenon (interaction radius, reaction/activation times and probabilities), we were able to reproduce the size, form, velocity and stability of the wave.

Figure 2b shows the probability of generating a wave when a small group of varying size attempts to trigger it under different values of the excitation threshold. Our results indicate that the dependence of the eventual occurrence of a wave on the number of initiators is a sharply changing function — that is, triggering a Mexican wave requires a critical mass of initiators.

Our approach may have implications for

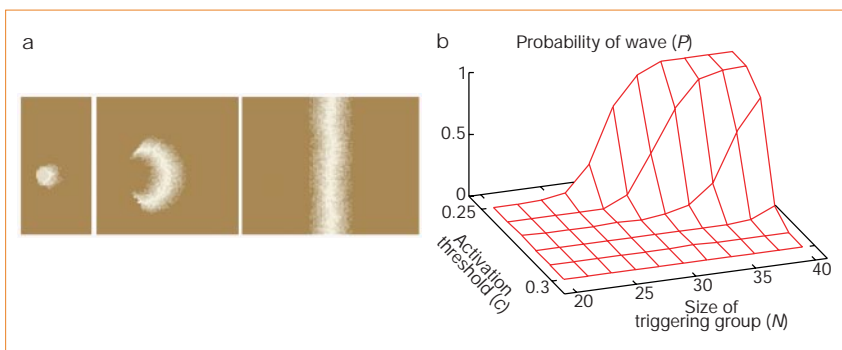


Figure 2 A model of the Mexican wave. If the weighted concentration of active people within a radius R around a person is above the person's threshold, c_i (chosen randomly from $[c - \Delta c, c + \Delta c]$), then that person becomes activated. Weights decrease exponentially with distance and change linearly with the cosine of the direction, so that people on the left of an individual have an influence that is w_0 times stronger than those on the right. The direction of the wave's motion is determined by this anisotropy because of the breakdown of spontaneous symmetry in the early stages as a result of anticipation and anisotropic perception (as most people are right-handed). **a**, Snapshots of the n -state model in which, after activation, a person deterministically goes through n_a active states (stages of standing up) and n_r refractory states. The wave is shown at 0.5 s, 2 s and 15 s after the triggering event, in a crowd with 80 rows of seats; brightness corresponds to the level of activity. Parameters are $n_a = n_r = 5$, $c = 0.25$, $\Delta c = 0.05$, $R = 3$ and $w_0 = 0.5$. **b**, The ratio of successful triggering events, $P(N, c)$, where P is the probability of a wave occurring as a function of the number of people, N , in the group who are trying to induce a wave, and the average threshold, c . Parameters are as in **a**; data represent means from 128 simulations.

crowd control. For example, in violent street incidents associated with demonstrations or sporting events, it is essential to understand the conditions under which small groups can gain control of the crowd, and how rapidly and in what form this perturbation or transition in behaviour could spread.

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Metallurgy

High nickel release from 1- and 2-euro coins

The amount of nickel is regulated in European products that come into direct and prolonged contact with human skin¹ because this metal may cause contact allergy, particularly hand eczema^{2–4}. Here we show that 1- and 2-euro coins induce positive skin-test reactions in sensitized individuals and release 240–320-fold more nickel than is allowed under the European Union Nickel Directive. A factor contributing to this high release of nickel is corrosion due to the bimetallic structure of these coins, which generates a galvanic potential of 30–40 mV in human sweat.

We performed skin tests with 1- and 2-euro coins in seven patients known to have nickel-contact allergy. After 48 and 72 h with these coins fixed by transparent tape onto their skin, all seven patients showed a strong reaction, with erythema, infiltration and formation of vesicles; they showed no reaction to 1% zinc chloride in Vaseline or to 1% copper sulphate in water.

In a quantitative nickel-release test (the European Standard EN 1811; ref. 5), the 50-cent coin did not release a measurable amount of nickel, as expected. However, we found that the 1- and 2-euro coins released more nickel than pure nickel itself (Fig. 1).

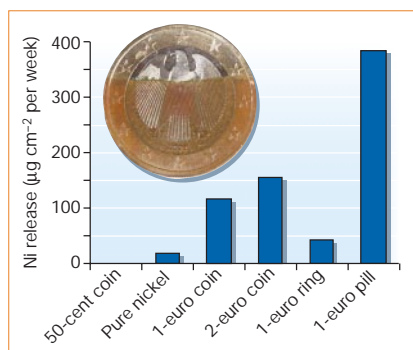


Figure 1 Release of nickel from euro coinage compared with that from pure nickel in artificial sweat, as measured by the EN 1811 standard reference test⁵ (values here have not been divided by 10, as specified for the test). Release of nickel from the bimetallic 1- and 2-euro coins is higher than from pure nickel. Inset, corrosion of a 1-euro piece after partial immersion in artificial sweat for 36 h.

This was particularly high from the inner component (the ‘pill’) of the 1-euro coin, but not for the outer component (the ‘ring’). These values are among the highest nickel-release rates ever measured on coins (see refs 6, 7 for a comparison).

In the 1-euro coin, the ring is made of a yellow alloy (‘nickel brass’) that consists of copper with 20% zinc and 5% nickel by weight; the white (‘cupro-nickel’) pill is copper with 25% nickel by weight; in the 2-euro coin the ring is cupro-nickel and the pill is nickel brass. As 1- and 2-euro coins are bimetallic, we measured the galvanic potential between the two metals with a high-impedance voltmeter after mechanically separating the pill and ring of a freshly minted coin and immersing them in either artificial sweat or saturated NaCl solution.

We found a difference in electrode potential between the two metals (the yellow metal was more negative and the white more positive) that was dependent on time and on the electrical resistance of the connector. After immersion for 24 h at ambient temperature in either solution, the potential difference between the two alloys stabilized at 40 mV (it was about 30 mV during the first 10 hours, then drifted slowly upwards) for a resistance of 100 kΩ.

It is well known that a current can enhance galvanic corrosion and thereby cause more nickel release. In thin irregular electrolyte layers such as sweat deposits, galvanic corrosion should occur primarily near the bimetallic junction because of the high resistance to lateral current flow in the thin layer. We measured electrochemically the relative rates of corrosion of each alloy, and found that the yellow metal dissolves at least five times faster than the white in the active-corrosion range (results not shown). Therefore, although the yellow component contains only one-fifth of the nickel of the white, its rate of nickel release is as high as that from the white component, or possibly higher, because the contact areas of the two alloys with the skin are about the same.

Corrosion of the 1-euro coin is visible after immersion for 36 hours in artificial human sweat: the colours changed to brown and the surface structure was damaged (Fig. 1, inset). No corrosion is evident, however, in a Swiss 1-franc coin, which consists of 25% nickel and 75% copper,

under these conditions (results not shown).

We conclude that the actual release of nickel from the present 1- and 2-euro coins exceeds the value acceptable for prolonged contact with human skin (as defined by European Union directive 94/27; ref. 1) by a factor of between 240 and 320 (Fig. 1). Whether or not this is acceptable by European standards hinges on the meaning of ‘prolonged’ contact. Further investigation is warranted not only into the epidemiological implications of such high-nickel-releasing coins but also into the factors that promote nickel release, such as the crevice between the pill and the ring — a potential corrosion site.

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Gene therapy

Biological pacemaker created by gene transfer

The pacemaker cells of the heart initiate the heartbeat, sustain the circulation, and dictate the rate and rhythm of cardiac contraction¹. Circulatory collapse ensues when these specialized cells are damaged by disease, a situation that currently necessitates the implantation of an electronic pacemaker². Here we report the use of viral gene transfer to convert quiescent heart-muscle cells into pacemaker cells, and the successful generation of spontaneous, rhythmic electrical activity in the ventricle *in vivo*. Our results indicate that genetically engineered pacemakers could be developed as a possible alternative to implantable electronic devices.

In the early embryonic heart, each cell possesses intrinsic pacemaker activity. The mechanism of spontaneous beating in the early embryo is remarkably simple³ — the opening of L-type calcium channels causes depolarization, and the subsequent opening of transient outward potassium channels leads to repolarization. As development