PHYSIOLOGICAL TREMOR

A slight oscillation at approximately 10 cycles per second accompanies the normal contraction of voluntary muscle. It appears to be caused by a "hunting" mechanism in the reflex arc that controls the muscle

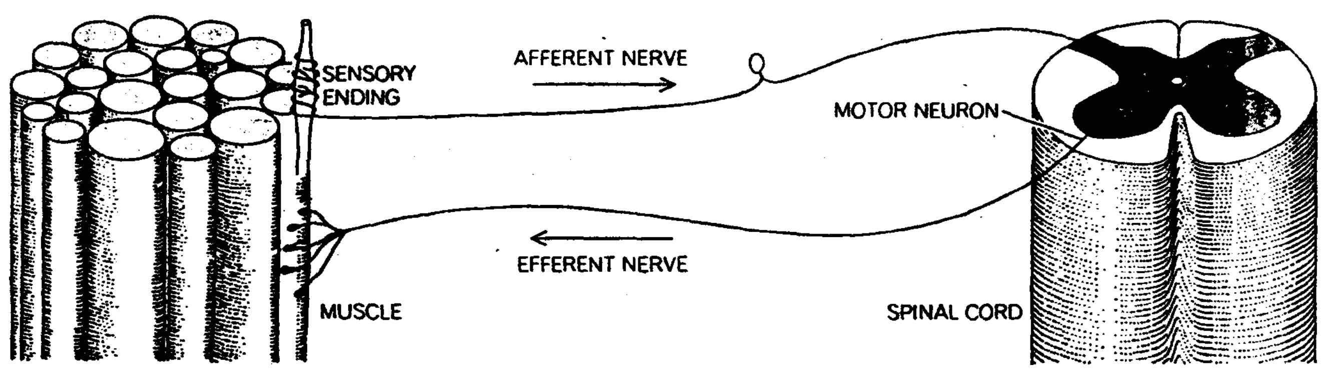
by Olof Lippold

The contraction of a voluntary muscle is accompanied by tremors of the muscle in the form of minute oscillations. In an electrical recording of the muscle's activity the tremor can be seen as a trace of very fine rhythmic movements superimposed on the record of the contraction itself. The amplitude of the oscillations is about a hundredth or a fiftieth as large as that of the total movement produced by the contraction, and the predominant frequency, in man, is about 10 cycles per second. This rippling of the muscle, known as physiological tremor, was discovered many years ago to be a normal accompaniment of the voluntary muscles' activity. Various explanations were proposed. There were theories that the factor generating the tremor might be the heartbeat or the alpha waves of the brain or activity of a group of synchronized motor neurons motor nerve cells—in the spinal cord or some form of local activity involving only the contracting muscle or its neural junction. All these theories fail to ac-count for the actual behavior of the tremor, as we shall see. It remained for a modern insight in technology, arising from the investigation of servomechanisms and the role of feedback, to suggest what now seems to be the correct explanation of physiological tremor.

The first clue leading to this explanation came from a study of muscle tremor made a little more than a decade ago by Martin Halliday and Joe Redfeam at the National Hospitals for Nervous Diseases in London. Applying the techniques of Fourier analysis to the frequency spectrum of physiological tremor, they found that the major part of the tremor consisted of an oscillation of the reflex mechanism that controls the length and tension of a stretched muscle. The fact that the tremor's spectrum peaks sharply in the narrow range between eight and 12 cycles per second then pointed to a likelihood that physiological tremor is an oscillation like the "hunting" behavior of mechanical servomechanisms, because characteristically in such systems the energy tends to be concentrated in a rather narrow bandwidth.

Consider the simple thermostat, a servomechanism designed to maintain temperature at a constant level. In this feedback control system there is an unavoidable delay in the response to a change in the temperature of the regulated space or object, so that the system overcorrects and the temperature keeps oscillating slightly above and below the desired value. In short, the temperature is continuously in a state of slow "tremor."

Built into the body of every mammal is an entire com4lex of biological servomechani, analogous to the thermostat, that operate to maintain the body's internal stability. Many of these Fhomeosta mechanisms are nervous reflexes. or Instance, there are reflexes that ad-just kite diameter of the eye pupil to control the amount of light impinging on the retina, there are nerve pathways



REFLEX ARC that senses and controls the stretching of a voluntary muscle consists of three main components: a sensory ending within the muscle that signals the amount by which it is stretched, an afferent nerve fiber that conveys the signal to the spinal cord and a motor neuron in the cord that in response sends a contras tionactivating signal to the muscle by way of the efferent nerve. Physiological tremor has been investigated in the author's laboratory at University College London by interrupting this feedback loop at a suitable point, by modifying the properties of the loop and by introducing various inputs into the opened or intact loop.

that keep the blood pressure constant and there are the stretch reflexes of muscle.

In the case of the stretch mechanism the control system is a reflex are consisting of three main components: a sensory ending within the muscle that signals the amount by which it is stretched, an afferent nerve fiber that conveys the signal to the spinal cord and a motor neuron in the cord that in response sends an activating signal to the muscle by way of the efferent nerve. A simple example of the operation of such an arc in maintaining stability is the reaction to a slight

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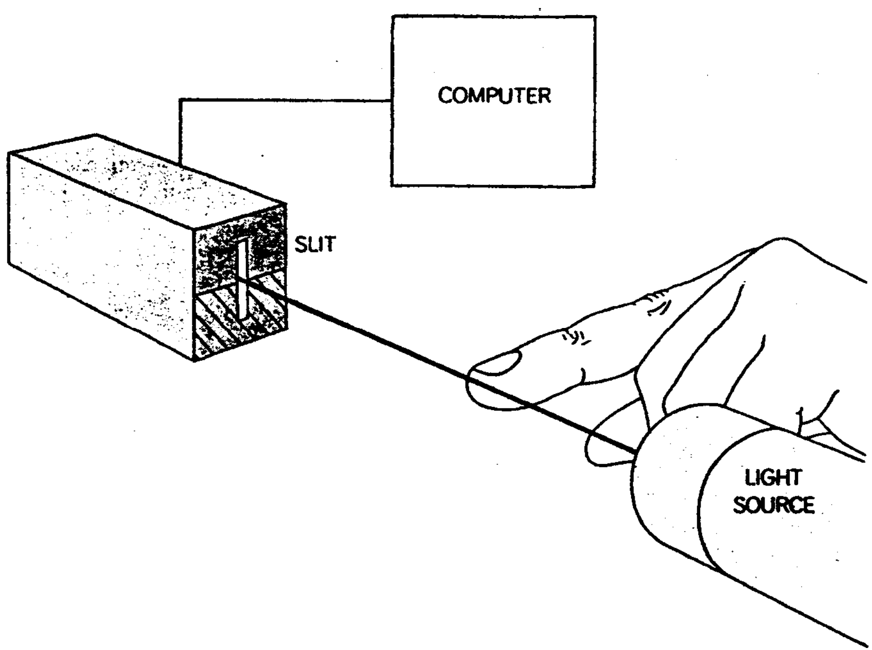
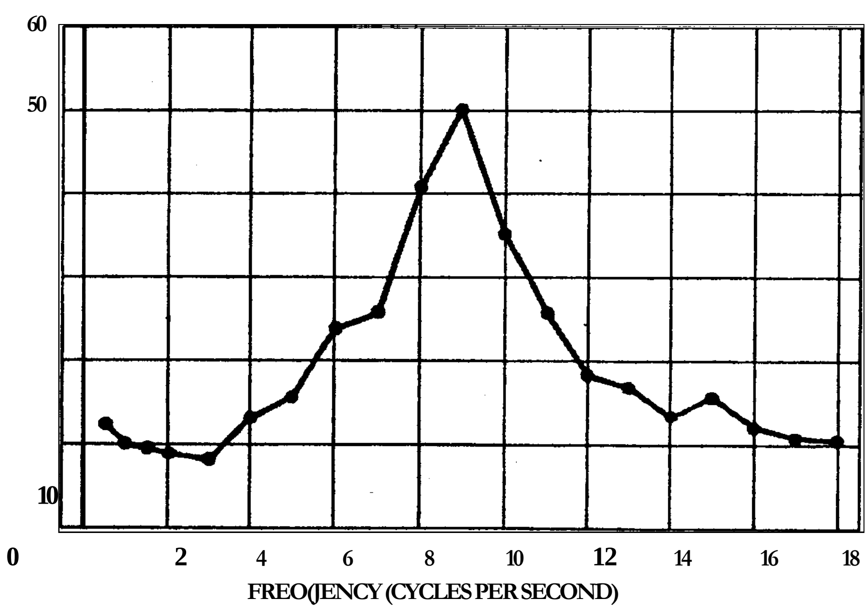


EXPERIMENTAL APPARATUS used by the author and his colleagues for recording physiological tremor in humans is shown in this illustration. The subject's forefinger interrupts a beam of parallel light falling on a ground-glass slit. Behind the slit is a photodetedor, the output of which is amplified and fed into a small fixed-program averaging computer.

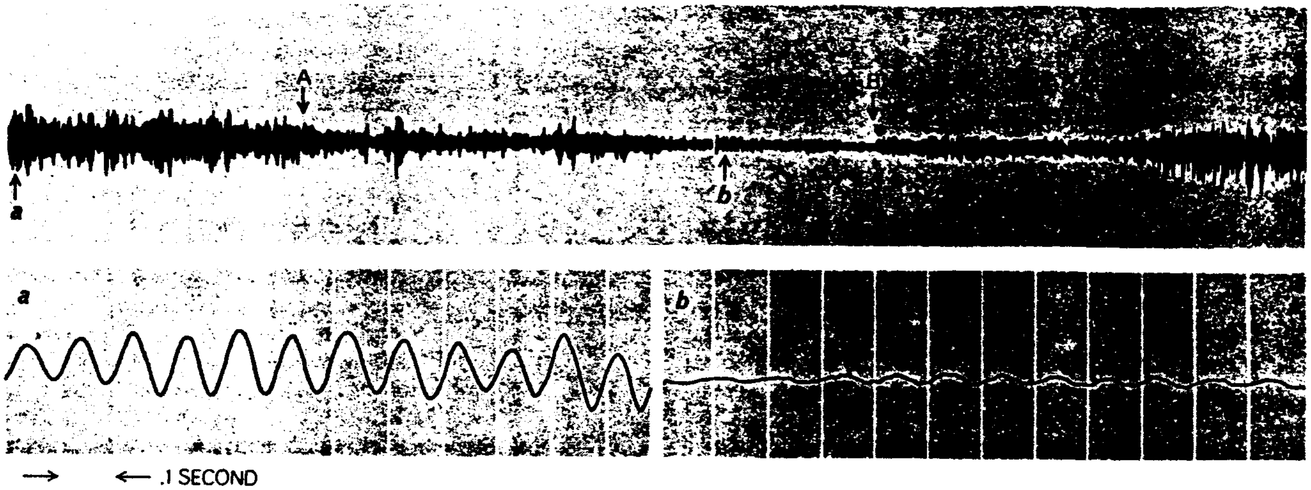


TYPICAL FREQUENCY SPECTRUM for finger tremor was measured using the apparatus depicted at the top of the page. The curve shows the acceleration of the finger plotted against the frequency of the oscillation in order to bring out the fact that finger tremor takes place In a dear-cut frequency band extending roughly from six to 12 cycles per second, superposed on a more or less flat base line. Since acceleration is proportional to the displacement of the finger times the frequency squared, the points on the ordinate were obtained bv multiplving the first differential of the outnut of the photodetector bv the freauencv.

push tending to tilt a standing person forward and thus move him off balance. The slight stretching of the extensor muscles in the back of the legs resulting from the push generates trains of nerve impulses that go to the spinal cord and there activate the appropriate motor neurons, which in turn incite a small strengthening of the contraction of the leg muscles, thus correcting the posture. All muscles are supplied with this kind of control system, usually operating in a more complex way than I have just described. The control generally is concerned either with damping the muscle's movements or with ensuring that for the given action of the operated limb there is an appropriate relation between the muscle's tension and its length.

Once it was realized that physiological tremor in muscle had much the same character as the oscillations of a mechanical control system, physiological tremor became accessible to the kind of detailed investigation in mathematical terms that engineers employ in studying the parameters of a servomechanism. The problem can be attacked experimentally by three devices: (1) opening, or interrupting, the feedback loop at a suitable point, (2) modifying the properties of the loop, (3) introducing various inputs, such as step functions (variables that change value in steps), into the opened or the intact loop. In our laboratory at University College London we have applied these methods to look into the causation, properties and behavior of tremor in muscles.

We began by examining the effects of interruption of the loop. Since cutting the loop on the efferent side would simply abolish all activity of the muscle under study, it was obvious that significant information could be obtained only by interrupting the afferent side. This could be done at the muscle spindle generating the sensory message, in the fiber conveying the message to the spinal cord or at the fiber's synapse with the motor neuron in the cord. It happens that certain patients are afflicted with an afferent interruption: tertiary syphilis of the central nervous system sometimes abolishes the conduction of impulses by the afferent nerve. We found that these patients generally showed no sign of the oscillation peak around 10 cycles per second, the mark of physiological tremor, when a muscle was stretched. Similarly, the tremor peak disappeared in cats when the afferent nerve was cut experimentally. The same result was obtained when we interfered with the normal ac-



TREMOR CAN BE REDUCED by cutting off the blood supply to the muscle. The top trace represents a forefinger tremor that gradwally disappears after a blood-pressure cuff is inflated at the time indicated by arrow A. After the cuff is deflated at arrow *B*, some two minutes later, the tremor gradually retntns. The two bottom traces, made on faster-moving recording paper at the times indicated by arrows a and b, show the detailed wave form of the tremor before and after the blood supply has been cut off.

tion of muscle spindles in signaling the stretching of the muscle. Cutting off the blood supply to a muscle causes profound disturbance of these spindles; they fire impulses spontaneously at a high rate and become almost completely unresponsive to stretching. When we cut down the blood supply to a muscle with an inflated cuff applying pressure to the artery, we found that physiological tremor in the muscle disappeared within 30 to 60 seconds. All in all, these observations and experiments tended to sup-port the hypothesis that physiological tremor is associated with muscular con-traction and signifies an oscillatory process taking place in the stretch-reflex loop.

Turning to the second device for experimental investigation—modification of a property of the loop—we selected first a treatment that might affect the oscillation frequency. In the stretch-reflex cycle there is a certain major period of delay: it consists in the time the muscle takes to contract after receiving the activating signal from the spinal cord and then to stretch again (either as a result of its elasticity or in response to a load on the muscle). Obviously if tremor is an oscillatory process, the frequency of the oscillations will be affected by any change in the duration of the delay. On this premise we performed the experiment of cooling or warming the muscle, which would be expected to lengthen or shorten the delay interval. The experiment in human subjects consisted in immersing a leg in water at a given temperature and recording the tremor frequency with a transducer *[see illustration on next page].* Most subjects were able to stand temperatures up to about 113 degrees Fahrenheit and down to the freezing temperature of 32 degrees, but we must admit that one of our best subjects fainted when he put his leg in really cold water!

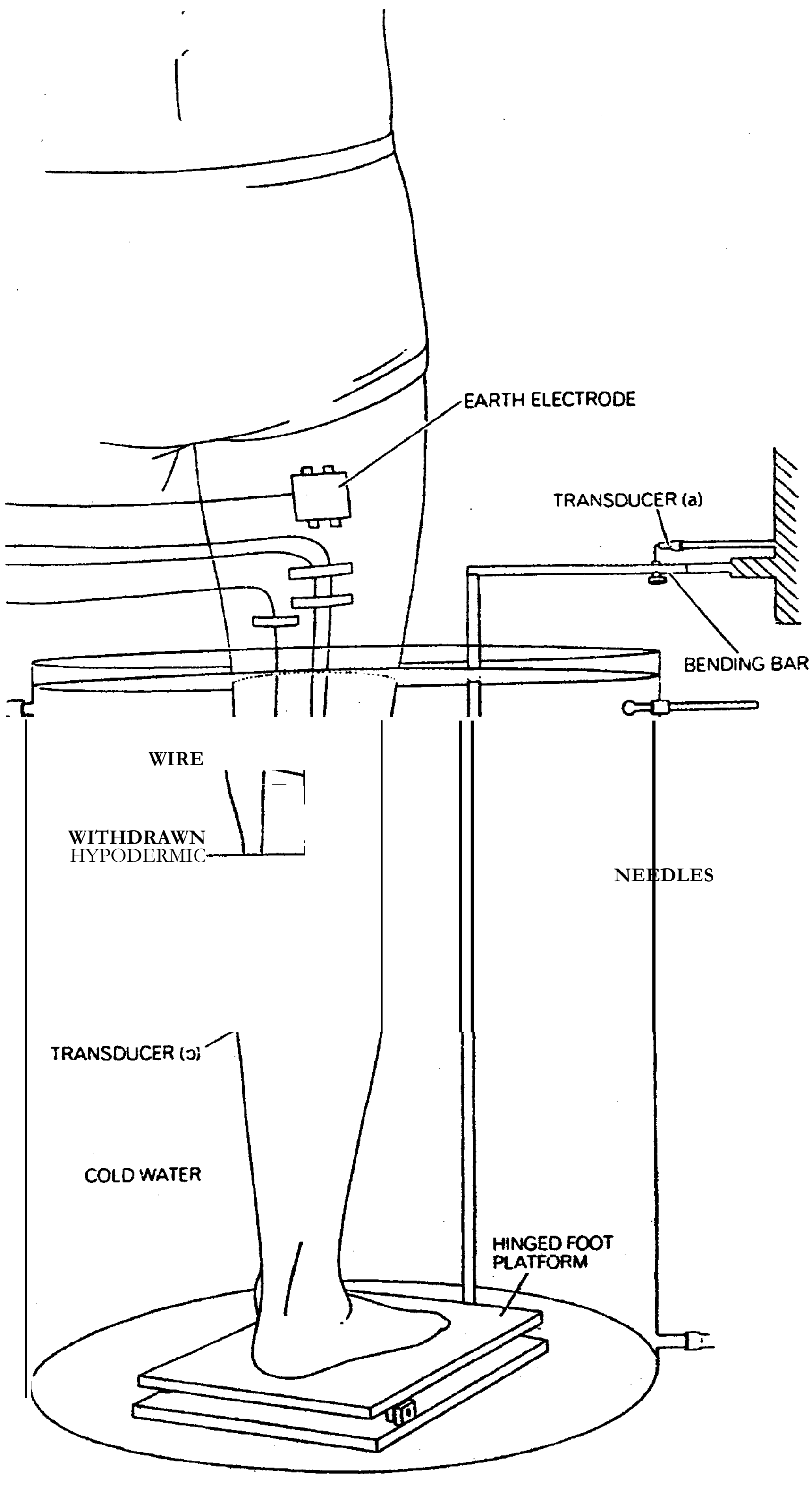
We found that cooling could slow the tremor from the normal rate of about 10 cycles per second to as low as five cycles, and rewarming could speed it up to *nearly* 15 cycles per second. It seems safe to conclude that the only reasonable interpretation of these results is that the tremor is indeed the kind of oscillation that takes place in a servo loop. We found also that in trembling muscle groups of action potentials accompanied the tremor and remained in phase with it as the oscillations slowed down or speeded up. The presence of the action potentials strongly indicates that the oscillations actually represent nerve-generated, synchronized muscle activity rather than, say, dying mechanical reverberations of the trembling limb.

Just as the tremor *frequency* could be changed by altering the delay interval, it might be expected that the tremor *amplitude* could be changed by modifying the system's sensitivity, or what engineers call the "loop gain." The signal-generating spindles of muscle offered an opportunity for such modification. These spindles have a complex structure, typically consisting of a central bag that contains the stretch-sensitive nerve endings and muscle fibers that contract and pull on the nuclear bag when they are activated. The fibers have their own activating nerve supply, provided by small motor fibers called "gamma" nerves because of their small diameter. Consequently some experimental intervention that activated the gamma nerves should increase the firing rate of the muscle spindles for a given stretch of the main muscle, thus enhancing the sensitivity of the entire reflex arc.

A simple maneuver called "Jendrassik's reinforcement" is known to produce a general enhancement of reflex action by way of activation of the muscle spindles' motor fibers. The maneuver consists in gripping one hand with the other and holding on while attempting to pull them .apa[im]rt. Applying this test, we obtained a most gratifying increase in the overall amplitude of physiological tremor. 1This again supported the oscillation theory.

For the third test of the theory—introduring inputs into the loop—we injected perturbations, by means of a mechanical prod, into the loop in an intact condition. As the prod we used a moving-coil transducer (driven by a power amplifier) that vibrated with a constant amplitude of two millimeters and prodded a human subject's extended middle finger. In order to record the tremors induced in the finger, it was placed in the path of a parallel beam of light directed at a photodetector mounted behind a vertical slit of ground glass; any oscillations of the finger were thus focused and measured *[see top illustration on page* 8].

It turned out that a prod of the freely extended finger lasting 30 milliseconds usually generated a series of five or more roughly sinusoidal waves. The frequency of the waves was the same as that of nor-



LEG MUSCLES WERE COOLED in this experimental apparatus designed to measure the effect of temperature on the frequency of a muscle's tremor. Copper electrodes were inserted into the muscle through hypodermic needles, which were subsequently withdrawn as shown. A transducer was employed in two alternative positions to measure the muscle's "twitch" time. In position a the transducer was attached to a bending bar connected to a hinged foot platform; in position b it was strapped to the calf and connected to a needle inserted through the skin with its tip in the muscle. Water at the desired temperature flowed through the tank, while the subject stood upright in as normal a posture as possible. Reflexes were elicited by means of sudden blows on a steel strip attached to the foot plat-form, raising the platform abruptly. The tension in the strip was recorded during maneuver.

mat tremor (generally about 10 cycles per second), and the amplitude was roughly uniform in a given subject. This could be taken as evidence that the waves were phase-locked to the mechanical input and hence represented oscillations such as would be expected in an underdamped (that is, flexible) servo-mechanism. Superimposed on the 10-cycles-per-second waves we often found, in the early part of the record, vibrations of about 25 to 30 cycles per second. These waves proved to be due to the mechanical, die-away resonance of the finger, as was shown by the fact that they were retarded when a load was placed on the finger.

Was the 10-cycles-per-second oscillation purely mechanical in nature or did it have the same neurological origin as physiological tremor? Several items of evidence indicated that it was in fact nerve-generated. Its frequency was always the same as that of the customary physiological tremor in any given individual. Warming or cooling the muscle or cutting off the blood supply produced the same results as when these treatments were applied to physiological tremor. Bursts of action potentials, phase-locked with the oscillations, took place in the prodded finger as they do in normal tremor, and the amplitude of the oscillations and the size of the action potentials tended to increase rather than to die away, indicating that energy was somehow being fed into the system and thus giving further support to the idea that the prod had initiated a process involving nerve activity.

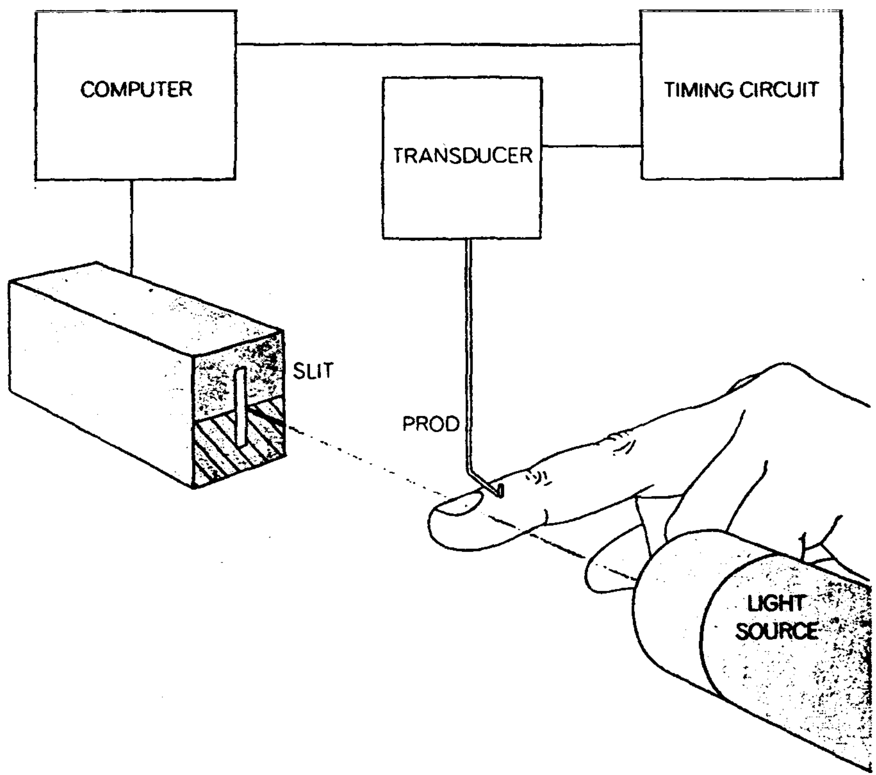
With the prodding technique we obtained new evidence that the waves of normal tremor represent oscillations in a self-sustaining feedback loop. It would be expected that in such a system the injection of an input of the same amplitude as that of the tremor would either enhance or suppress the tremor waves, depending on whether it was applied in phase or out of phase with those waves. A prod less than half a millimeter in amplitude could serve as such an input. We carried out this experiment in various ways. In one version we applied the prod at random intervals while the muscle was in continuous tremor. Then, examining the record, we could see what happened to the oscillation when the in-put occurred at various phases of the wave form. This experiment showed that the tremor was indeed enhanced when the prod occurred in phase and was suppressed for several cycles when the prod came in anaphase ***(see*** **bottom** ***illustration on page 8).***

Experiments in stimulating an isolated nerve-muscle preparation at various rates show that when the rate of stimulation is raised to above 10 cycles per second, the individual twitches fuse, or merge, into a more or less smooth tetanus. Similar experiments in stimulating a human muscle **through** the skin with **brief electric shocks produce the same result. It is also known that the motor units in muscle do not often fire at much below the 10-cycles-per-second frequency. Thus, assuming that the units fire more or less at random, the result of the filtering effect would be that only the action potentials with a frequency in the neighborhood** of 10 cycles per second (in a normal human subject) would be effective in producing the small discrete **contractions** that account for the major part of tremor.

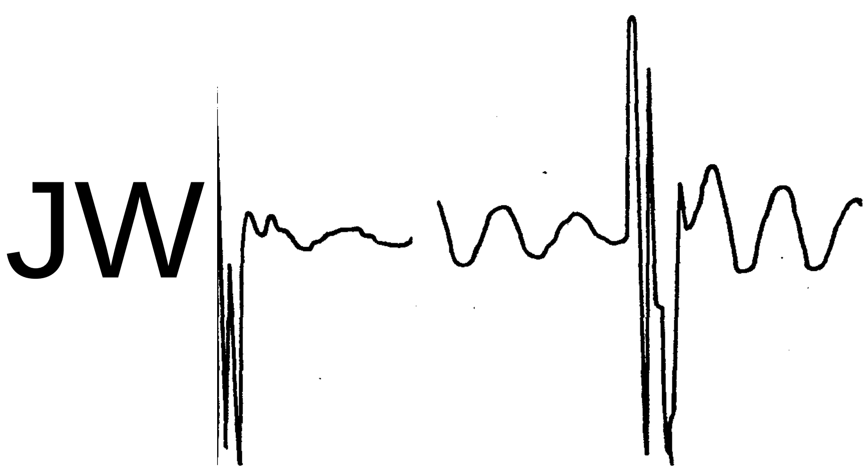
There are a number of powerful objections to this theory, however. In the first place, if **the motor** units fire **randomly, there will be no discernible** tremor, even if all the units are firing at **the rate** of **about** 10 cycles per second, **unless there is a synchronizing mecha**nism **that causes** them **to** fire in **phase. Secondly,** the **theory** implies that **a** factor **such** as **fatigue,** which **considerably lowers the threshold frequency** at which **individual** twitches merge, **should** slow **the tremor frequency** to much below 10 cycles per second; **actually** it is found **that during and** after fatiguing contractions a muscle **always** shows a slight rise in **the** tremor **frequency and a** marked **increase in its amplitude.**

The results of some of the experiments I have mentioned are also not compatible with the filter **theory.** It cannot **explain the clear correspondence** be**tween the grouping of action potentials and the** tremor **waves, nor** the fact **that a brief displacement of a stretched finger by a prod produces a train of waves phase-locked to this perturbation. More**-over, **when an isolated muscle** is stimulated **through** its motor nerve with re**peated** shocks at rising frequency, the muscle's contraction shows no increase in tremor at **about** 10 cycles per second, as **would be expected** if there were a filtering effect.

If we accept the **hypothesis** that the **physiological** tremor at from eight to 12 cycles per second arises as a hunting **phenomenon** in the stretch reflex arc, in the final **analysis** we must ask why this **biological servomechanism** is so "inefficient" as to allow tremor. Most man-**made control mechanisms are** provided **with devices that automatically suppress oscillation and thus ensure** stability. An**alyzing the control** system in the reflex arc, **however,** we can see that excessive **damping** in this system would inevitably slow **the response to** stimulation. Reflex **action therefore** must **involve** a necessary compromise between speed of re**sponse on the one hand and a certain degree of overshooting,** or inaccuracy, **on the other.** We **have** found in exam**ining a large number of normal human subjects that** most **of** them have some **tremor superimposed on their muscular activity.** The **amplitude** of the tremor **usually does** not exceed **2** percent of the **physiological range of movement of a**



FINGER-PRODDING APPARATUS **employs a moving-coil transducer driven by a power amplifier that is fed with step-function signals from a crystal-controlled timing circuit. The timing circuit also triggers the averaging computer, which then derives the algebraical sum of the wave form of the finger displacements for a given number of consecutive prods.**



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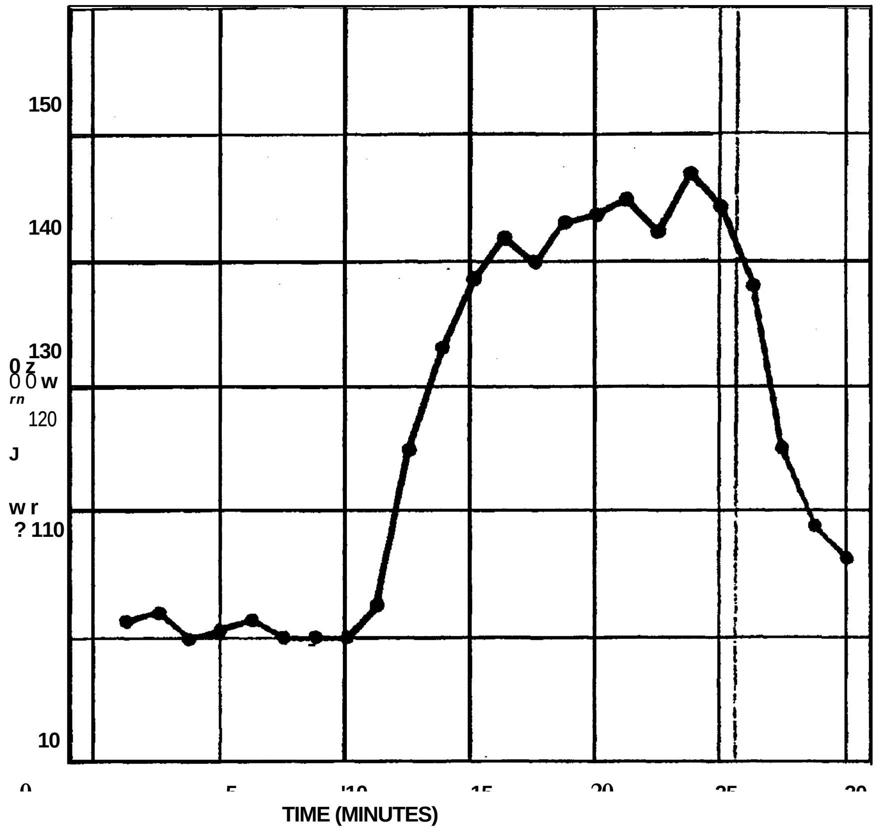
TREMOR IS SUPPRESSED OR ENHANCED **depending on the instant at which the prod is administered. When the prod is given out of phase with the ongoing tremor (left), the tremor is suppressed. When the prod is in phase *(right)*, subsequent waves are enhanced.**

In interpreting the results of all these studies we must first be clear about exactly what we mean by physiological tremor. Tremor in a muscle can be produced by a great variety of stimuli: the beating of the pulse, general movements involving a number of other muscles, even vibrations created by passing traffic. In identifying and examining normal physiological tremor, however, we are concerned specifically with the ripple that is superimposed on the voluntary contraction of a particular muscle and arises solely from this activity. Consequently in order to investigate physiological tremor in a finger, say, we must fixate the limb as a whole so that extraneous movements are excluded and we can focus on the indigenous oscillations of the finger muscle itself. One must also distinguish between normal physiological tremor and pathological tremor such as that of Parkinson's disease. The Parkinson tremor has a different frequency, peaking at about five cycles per second. Apparently, like normal physiological tremor, it is characterized by oscillating loops, but its origin is different, it probably involves the brain stem, and the experimental findings in the normal case do not necessarily have any application to the pathological one or vice versa.



EFFECT OF COOLING on tremor in leg muscle is indicated by this graph, which shows a large increase in the mean interval between bursts of action potentials in the calf muscle plotted during the course of the immersion of the leg in cold water. At the 10th minute the leg was cooled by filling the tank with water at a temperature of about zero degrees Celsius; the tank was emptied again at the 26th minute. Mechanical tremor waves recorded during this experiment bore a constant phase relationship with the action-potential bursts.

In the light of the new experimental information, how do the old theories about physiological tremor stand up? Let us consider first the theory of an origin in the brain. After the discovery by encephalography of the brain's alpha waves there were several reasons to suppose tremor might well be connected with those waves. The alpha rhythm is very similar to tremor oscillations in wave form and in frequency (ranging from eight to 13 cycles per second for alpha). In both cases the development of the frequency follows the same course, starting at five cycles per second in young children and accelerating to about 10 cycles in adults. Both alpha waves and tremor respond in much the same way to a sudden stimulus (such as a loud sound), to anesthesia, to sleep and to general alerting of the system. Yet the many attempts to find a causal connection between the two phenomena were fruitless. It became evident that the rhythms do not quite match each other, either in frequency or in phase. When the tremor frequency is changed drastically (for example by cooling the muscle), there is no change whatever in the brain's alpha rhythm. No delay interval such as exists in the tremor loop has been found in the brain's motor systems. Another finding arguing against the brain's involvement is the fact that when the spinal cord is severed from the brain, tremor at about 10 cycles per second can still be induced in muscle by stimulating the cord electrically.

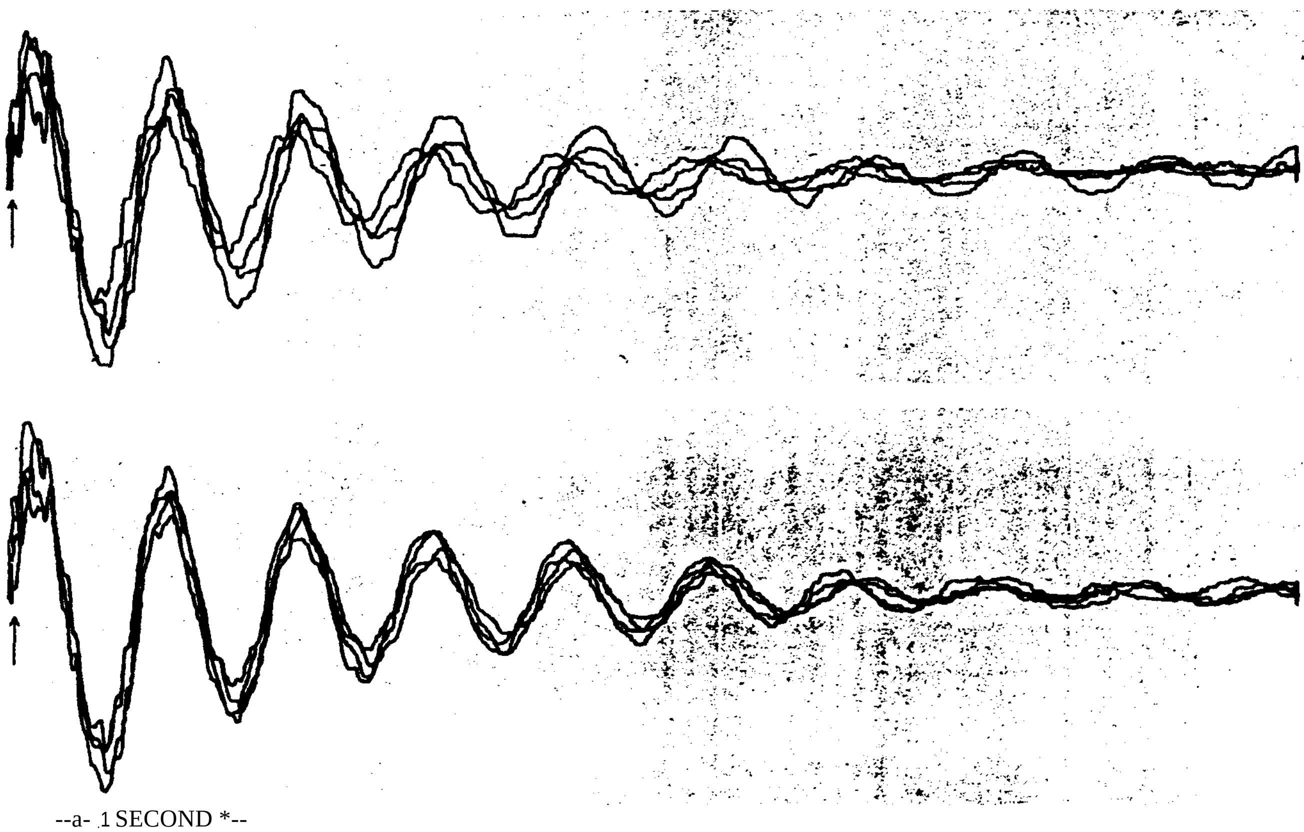
Although the search for a connection between physiological tremor and electrical activity in the brain was unsuccessful, it might profitably have been pursued further than it was. It now appears that the alpha waves may be generated by the extraocular muscles (voluntary muscles that control movements of the eyeball).

Then there is the simple and attractive theory that tremor may be originated by the spinal cord. This hypothesis has gone through several versions. One suggests that the group of motor neurons activating a muscle may somehow synchronize their discharge of impulses in a rhythmic fashion. The main problem with this theory is that it cannot explain how the cooling or warming of a muscle would alter the frequency of discharges in 4he cord, or how prodding a finger would generate tremor or bring about a change'in the amplitude of the oscillations: Another version of the theory argues that the rhythmic waves may be produced by a pacemaker of some kind in the cord, causing bursts of action potentials at about 10 cycles per second. Such systems have been found in the thalamus of the brain, but no evidence for the existence of such a center in the spinal cord has been established.

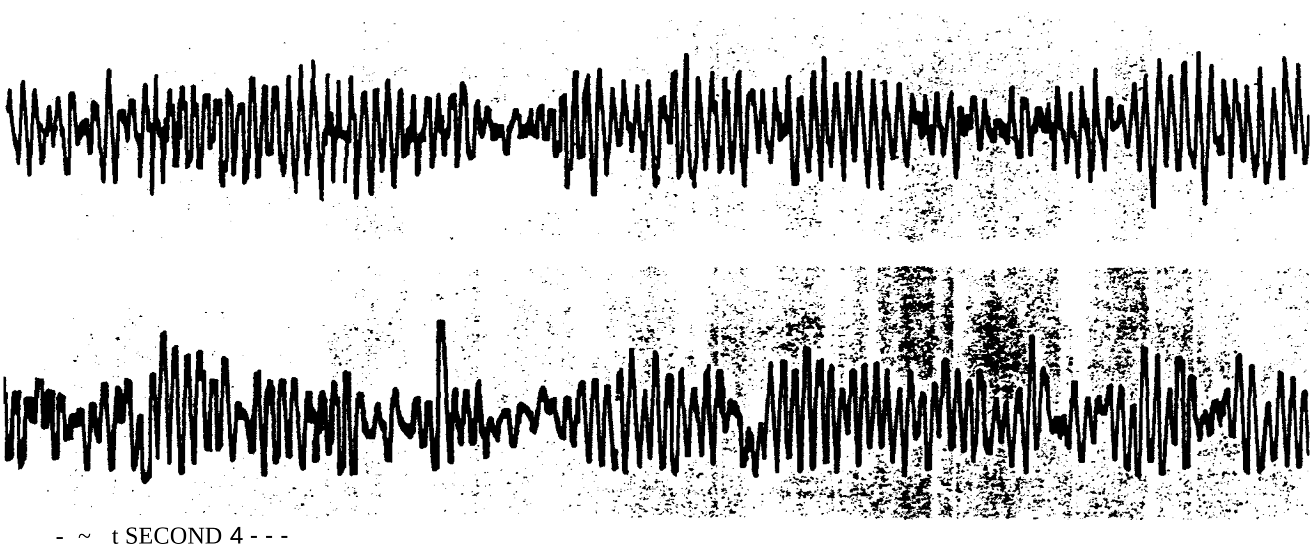
Finally, there is the hypothesis that tremor may be strictly a local phenomenon within the muscle (perhaps with participation by the neuromuscular junction), not involving the spinal cord or the brain at all. One ingenious and plausible idea imagines the muscle to be acting as a low-pass filter that screens out all impulses except those at about 10 cycles per second. We know that during a voluntary contraction motor units composed of functionally identical muscle fibers start discharging at about seven cycles per second and then accelerate up to 30 or 40 cycles per second as the strength of the contraction increases.

limb. It would appear that this amount of oscillation can be tolerated in the interest of maintaining a fast response.

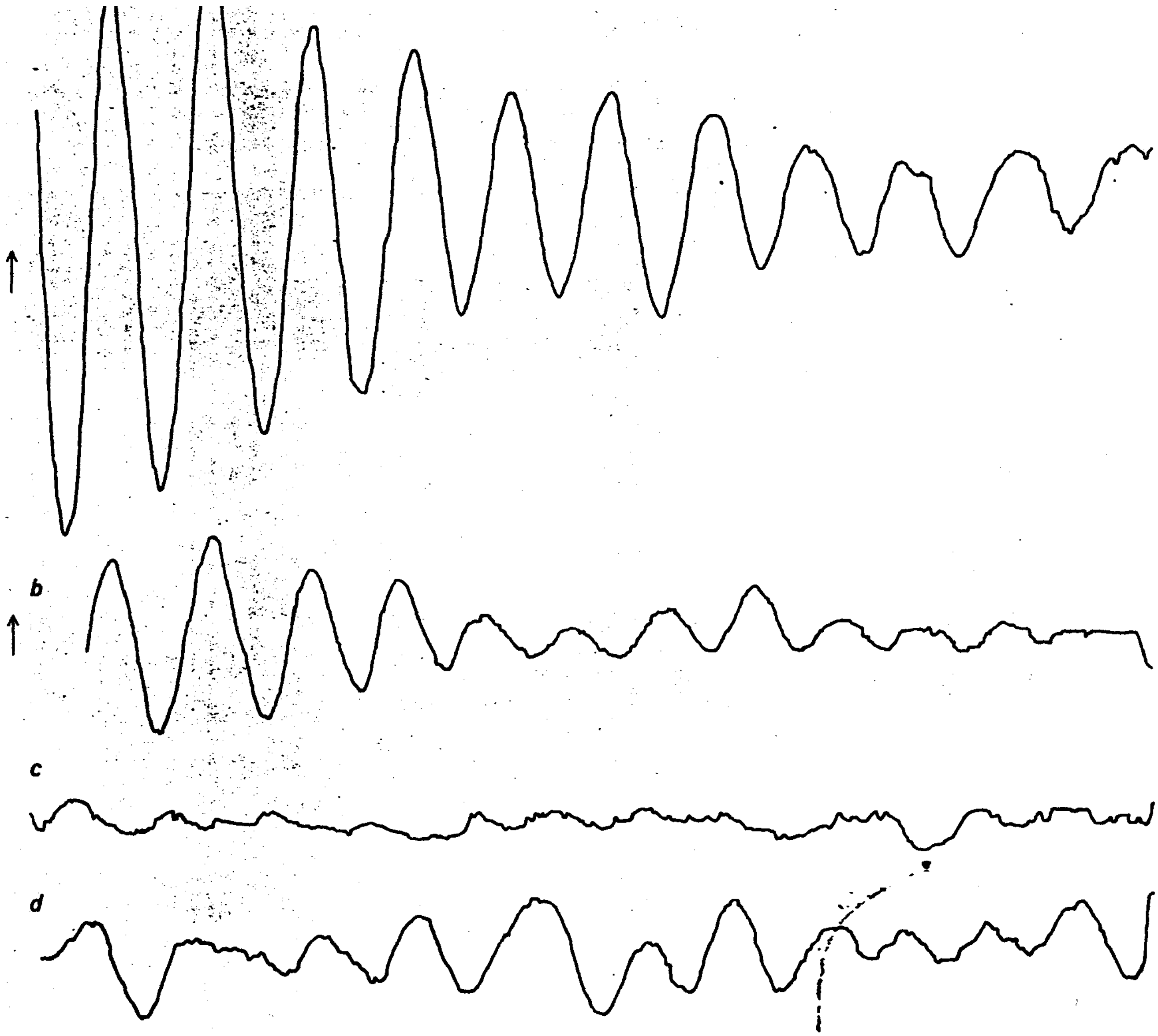
Why do some normal people have more tremor than others? We find that tremor varies not only from one person **to another but also in the same individual from time to time. Apparently the central nervous system normally brings about variations in tremor from minute to minute (leaving aside the changes that may be produced by factors such as fatigue, circulating adrenalin or pathological conditions such as hyperthyroidism).** We have already noted that Jcndrassik's maneuver can increase tremor, **presumably** because the strong contraction of a muscle results in a spreading "**irradiation**" of **nerve** action speeding up **the frequency** of **motor** impulses generally. **Some** recent experiments show that



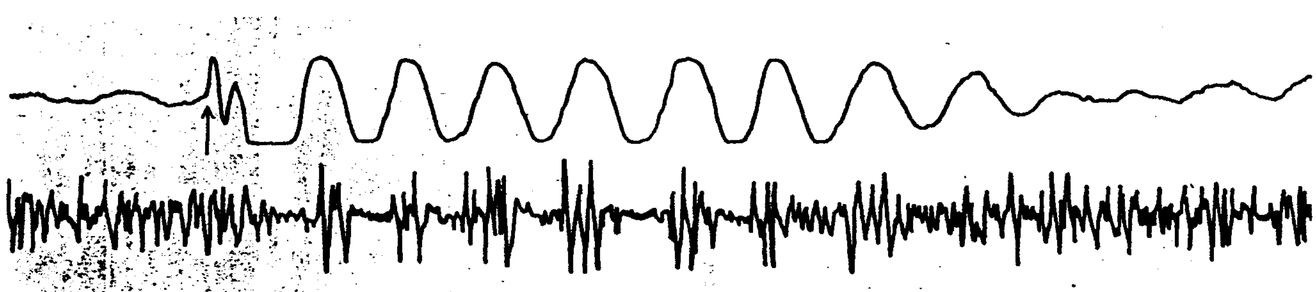
VISUAL FEEDBACK LOOPS appear to have little influence on either normal tremor or prod-initiated tremor. The superposed traces in both cases represent four sets of averaged responses to a forefinger prod given just before the arrow. The top traces were recorded with the eyes open, the bottom traces with the eyes closed. The recordings were made alternately with the same subject.



DECEPTIVE SIMILARITY is evident in these two traces, which were recorded simultaneously using the same subject. The top trace represents physiological tremor recorded in the forefinger. The bottom trace represents the "alpha" rhythm recorded from the scalp using a conventional electroencephalograph. Hypotheses relating these two types of oscillation have had to be abandoned.



SERIES OF DAMPED OSCILLATIONS can be induced in a trembling finger by means of a single prod. A brief prod delivered just before the arrows was responsible for the two upper traces, Trace a is an average of 16 consecutive sweeps; trace b is one sweep recorded at four times the gain. The two lower traces are records of normal tremor in the finger. Trace c is a control run with 16 sweeps with no prodding at the same gain as trace a; trace d is a single sweep, again without prodding, at four times the gain.



ELECTRICAL SIGNALS from a prodded finger muscle were recorded by means of surface electrodes applied to the skin over the muscle. The bursts of action potentials recorded in this way *(bottom trace)* turned out to be very nearly in phase with the oscillating displacement of the finger *(top trace)* that resulted from a single prod administered at the time indicated by the arrow.

normal tremor can also be reduced. Resting a limb for some hours (for example by putting an arm in a splint) will reduce the amplitude of tremor in all the muscles of the arm. This may involve the same mechanism that is affected by a strong contraction; inactivity may result in a decrease of activity in the fusimotor system (the system of gamma motor nerves in muscle that cause the muscle spindles to contract), whereas strong contraction stimulates increased activity in the system.

Quite by chance, while we were screening a large group of students for visual defects, we came on a surprising and fascinating discovery. It turned out that people with poor eyesight tended to show an unusually large amount of finger tremor! This was particularly true of farsighted individuals: the amplitude of their finger tremor was much larger than normal. Tentatively we postulate that the explanation may be essentially the same as in the case of Jendrassik's maneuver. In farsighted people there is a conflict between the acts of accommodation (focusing the eyes on a near object, for example) and convergence (directing the optical axes of the eyes toward the object). Anatomically these two acts are joined, so that they cannot be performed separately; hence in a farsighted person any effort exerted to accommodate the lens to bring an object into focus also will tend to make the eyes converge on too close a point and thus produce double vision. A possible action to correct this might bring into play the rectus muscles of the eyeball that rotate the *eyes* outward horizontally. 'tVe can suppose, then, that the activity of these muscles may be intense enough to bring about stimulation of the fusiniotor systems and hence tremor in all the other muscles of the body while the individual is actively observing his environment. We have one piece of concrete evidence that this theory represents something close to the actual state of affairs. It is found experimentally that if a person with normal vision wears special glasses that make him functionally farsighted, after a day or so the amplitude of his finger tremor increases considerably.

Clearly physiological tremor is introducing us to a fascinating field of study. With the sophisticated techniques of investigation and analysis now available, there is reason to hope that we shall be able to find answers to some puzzles about normal muscle activity and also to learn something about how pathological tremors arise, thereby making them more useful as a diagnostic and prognostic tool in psychiatry.

The Author

OLOF LIPPOLD is reader in physiology at University College London. He qualified in medicine at University College Hospital in London in 1946, later serving there as tutor to medical students and as subdean of the faculty of medicine. His interest in brain function has led him to develop, among other things, a new method of psychiatric treatment. "We were investigating the action of small polarizing currents on the human brain," he writes, "and it was thought by our subjects, most of whom were mildly depressed, that the procedure had beneficial effects. We soon found ourselves running a small clinic for treating these people." A double-blind trial showed that there were indeed small but significant effects. Lippold, who advocates re-search in methods of teaching, has experimented with programmed learning.

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