great flexibility, and the illustration and practice of wiring are greatly simplified.

Three individuals can also be identified by using ferromagnetic sensing. The soft-iron collar of the third animal should weigh about 1 g. The excitation voltage to the sensor bridge is made reducible with a normally shorted series resistor, so that when it is in series, the amplified just fails to respond to the signal generated by the presence of the 0.6-g collar. Triggering of the normally wired amplifier by either collar now open-circuits the short and also establishes a pathway to a third recording channel. With the short open-circuited, only the presence of the heavy collar can trigger the amplifier. Thus, scoring of a passage by all three channels identifies the animal with the heavy collar; scoring by only two channels identifies the animal with the light collar; while scoring by only one channel identifies the third animal.

For identification of a fourth animal, conductance proximity sensing is employed in conjunction with the ferromagnetic method (see Fig. 1*A*). We have used the Bently D-151 detector (*3*), which is essentially an eddy current sensor. The head (*l* in Fig. 1*A*) of this unit contains a pancake-wound coil pick-off element which is loaded by the near approach of any conducting material (which appears to it as a shorted secondary coil). Loading of the pancake coil generates a change in the radio-frequency output of a regenerative radio-frequency oscillator (modified Colpitt configuration). The d-c envelope of this output is either monitored directly or converted to a digital signal by a "Schmidt trigger" binary switch. The collar of the fourth animal can be of any conducting nonferromagnetic material, but the dummy collar must then be nonconducting (*4*).

Note added in proof. It has come to my attention that T. Royama used magnetic proximity sensing in a nest recorder to differentiate between the visits of a male and a metal-banded female great tit, *Parus major* [*Brit. Birds* **52**, 295 (1959)].

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Taxonomic Status of the Pygmy Marmoset (Callithrix pygmaea Spix)

In an earlier issue of *Science* (*1*) there appeared a report on the pygmy marmoset as an experimental animal. Because it is possible that this species may become an established laboratory animal, it appears essential that its taxonomic status be correctly determined.

Callithrix pygmaea Spix is a form of the group of marmosets of which the brush-eared marmoset known as *Callithrix jacchus* Linnaeus is typical and of which it is the westernmost representative. This is a true pygmy. It does not deserve generic or even subgeneric rank. The original specimen (*2*) was collected by the German zoologist J. B. von Spix at Tabatinga (now Sapurara) on the north bank of the Amazon River on the Brazilian side of the Brazil-Colombian border, about 250 miles down river from Iquitos in the province of Loreto, Peru, from which area the stock now kept at Los Angeles was derived. It also occurs in the forested area on both sides of the upper Amazon and is known to be common in the area of the Napo, Copataza, and Pastaza rivers in northern Peru and the Oriente Province of Ecuador and along the lower Ucayali River, being on record eastward in Brazil at least as far as the Juruá River (Eirunepé, formerly João Pessoa). The type locality of Lönnberg's *niveiventris* (*3*) is very slightly further east, in the area of the mouth of the Teffé River (Lago de Ipesuna). There is no doubt that this is not different from the original *pygmaea* and that *niveiventris* is not a valid name. The Los Angeles material comes from an area clearly within the range of the original *pygmaea*, west, not east, of the type locality.

These conclusions have been recently confirmed by examination of a series of specimens from localities covering the whole area of distribution of this form, at the British Museum (Natural History), London, England.

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24 July 1961

Carotid and Vagal Afferents and Drug Action on Transcallosally Evoked Cortical Potentials

Abstract. The use of transcallosally evoked cortical potentials to study the action of intracarotidly injected drugs on cerebral synapses has necessitated the demonstration that vagal, baroreceptor, and chemoreceptor influences do not play essential roles in the drug effects observed —for example, the cerebral synaptic inhibitory action of serotonin.

Transcallosally evoked cortical potentials have proved to be very useful tools in studying the effects of drugs on cerebral synaptic function (*1*). In order to obtain central effects with little complication from peripheral ones the drugs have been administered by close arterial injection (injected "intracarotidly"), because this achieves adequate concentration in the ipsilateral hemisphere and the subsequent dilution in systemic blood ordinarily lowers the concentration to a subthreshold level for peripheral effects. Afferent inflow has been reduced by light anesthesia, or curarization (*2*) has been used to eliminate proprioceptive inflow. Since such isolation from the periphery is incomplete, we needed to examine the influence of persisting afferent inflows, such as inflows over cranial nerves, as in the case of the vagus; and, especially because of the comparatively high concentration of drugs bathing the carotid sinus and carotid body due to the intracarotid route of administration, we needed to examine the possibility of baroreceptor and chemoreceptor influences on the transcallosally evoked potentials.

Section of the vagus nerve in the neck, surprisingly, impaired the ability of serotonin to reduce the transcallosally evoked cortical potentials, but, as seen in cat experiments (Fig. 1), this was a temporary, reversible effect. It developed that actual section of the vagus nerve was not necessary, but that a crush would elicit the same temporary hindrance of cerebral serotonin action. Since recovery could be accelerated by subsequent application of cocaine to the cut end or to the crushed region (Fig. 1), and since prior application of cocaine prevented occurrence of the phenomenon, it is concluded that the interference with cerebral serotonin action by vagus section or crush does not imply dependence of serotonin's cerebral synaptic inhibitory

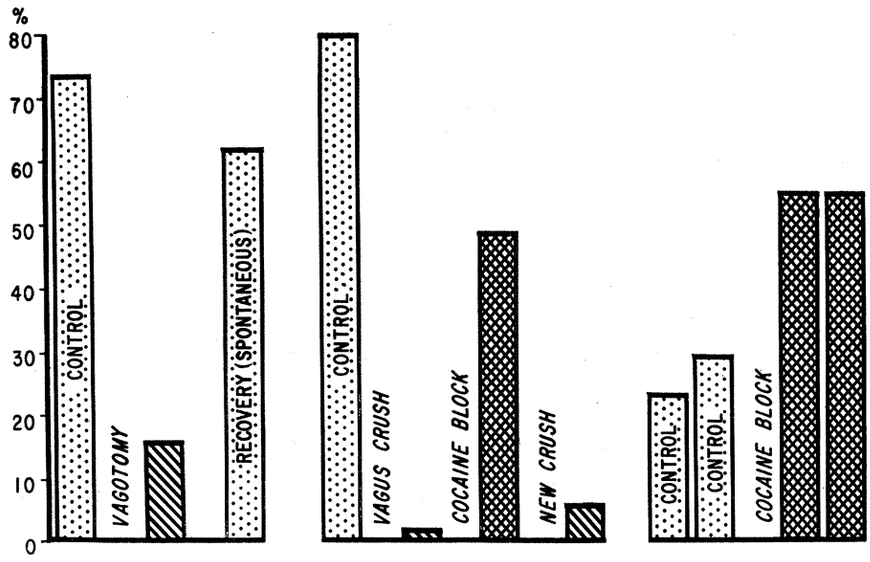

Fig. 1. Influence of vagal afferents on cerebral synaptic inhibition by serotonin.

action on intact vagi, but implies simply that the cut or crush initiates a strong enough volley of afferent impulses to complicate and obscure the central effects of serotonin.

In Fig. 1, the bar marked "control" indicates the percentage of inhibition by serotonin before section or crush. Spontaneous recovery ("healing of the killed end" or sealing off of the irritated region) occurred in about 90 minutes, while cocaine-induced recovery took place in about 5 minutes. The new crush, after cocaine, was applied to a fresh section of the vagus, central to the site of previous cocainization. That the influence of vagal afferents is an underlying aspect of the situation and that spontaneous vagal afferent impulses can modify the effect studied is illustrated by the group of bars at the right of Fig. 1. These show that, in an experiment in which serotonin effects were relatively small in two consecutive trials, cocaine block of the otherwise untouched vagus enhanced the serotonin action. Presumably there had been sufficient afferent inflow in the undisturbed vagus to partially offset the serotonin action, whose full effect was released by blocking the vagal inflow with cocaine.

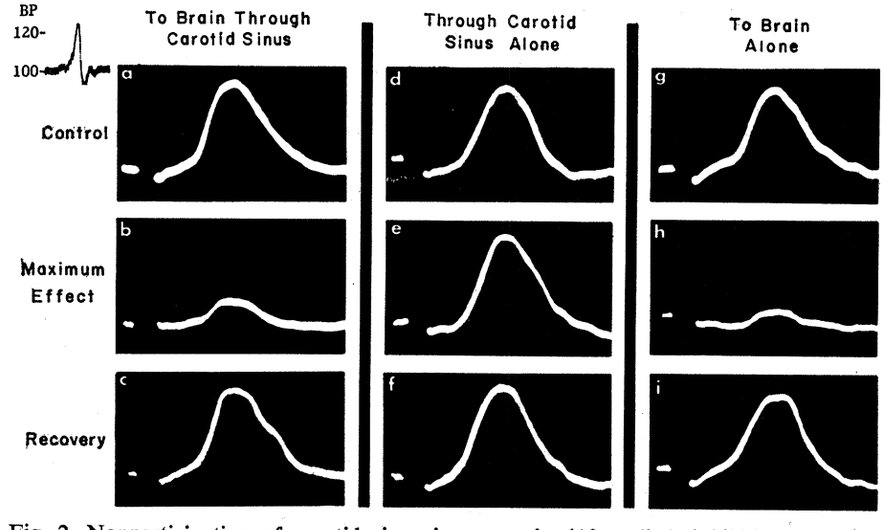

Fig. 2. Nonparticipation of carotid sinus in serotonin (10 μg/kg) inhibition of transcallosally evoked cortical potentials in a pentobarbitalized cat.

The possibility that intracarotid serotonin modulates baro- and chemoreceptor control, originating in the carotid sinus (*3*) and carotid body, thereby modulating the cortical potential, also required examination. First it was demonstrated that denervation of the carotid sinus, unilateral or bilateral, did not alter the cerebral synaptic inhibitory action of serotonin in these experiments. Secondly, it was shown (Fig. 2) that the cerebral synaptic inhibitory action of serotonin was not significantly altered when the carotid sinus and carotid body region were bypassed by injecting the serotonin cephalad to this region (compare *a, b, c* with *g, h, i*). Repeating the injection but allowing the serotonin to perfuse the carotid sinus and carotid body region alone, without access to the brain, had no effect on the evoked potential (*d, e, f*). Carotid reactivity is evidenced by the rise in blood pressure (Fig. 2, top left) that is induced by occlusion of the common carotid arteries below the sinuses.

These experiments indicate that in the lightly anesthetized cat the transcallosally evoked potential is still modifiable by afferent inflows, especially when they are as great as that initiated by trauma to the vagus nerve, but that the intracarotid administration of the doses of serotonin used does not produce baro- or chemoreceptor influences sufficient to modify the cerebral synaptic inhibition demonstrated by reduction in the height of transcallosally evoked cortical potentials. Completely isolated transcallosal and corticocortical preparations are under study (*4*).

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3 July 1961