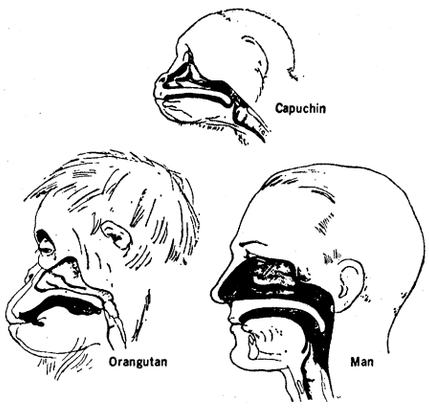

Fig. 4. Vocal tracts of monkey, ape, and man after Negus (*10*). The nonhuman primates lack a pharyngeal region where the root of the tongue forms a movable anterior wall. The nonhuman primates thus lack a speech-production mechanism where the area of the "back" pharyngeal region is variable and independent of the area of the "front" of the vocal tract.

by a high laryngeal impedance (*1*, *9*). We have also scaled up these formant frequencies to take account of the longer vocal tracts of these animals relative to rhesus monkey. The formant frequencies that would correspond to a uniform tube, 6.5 cm long, terminated at one end are also plotted in Fig. 2. The actual monkey and ape cries occupy only part of the vowel space of our computer-generated vowels. The only natural cry that is a signficant deviation from this schwa vowel is the chimpanzee cry which was produced by the animal with its lips rounded (*1*); the formant frequencies of this cry correspond most closely to configuration 7 of Fig. 1c, which represents the least rounded of our simulated rounded back vowels. Our computer-modeled configurations of the perturbed monkey vocal tract thus encompass and extend beyond the "acoustic vowel space" that was measured for actual utterances of nonhuman primates. The nonhuman primates previously recorded did not, in fact, use all of the articulatory maneuvers that we simulated for the rhesus monkey by means of the computer model.

The computer model further indicates that the possible acoustic vowel space of a monkey is quite restricted compared to the human range. Even if a rhesus monkey were able to manipulate his supralaryngeal vocal tract to make use of all of the possibilities that we considered in our computer model, he would not be able to produce the full range of human vowels. We can thus conclude that the vocal apparatus of the rhesus monkey is inherently incapable of producing the range of human speech.

In Fig. 3 we have presented schematized area functions for the human vowels /a/, /u/, and /i/ where we have approximated the vocal tract by means of uniform tubes for illustrative purposes. We have based these approximations on Fant's data (*3*). The supralaryngeal vocal tract can essentially be divided into an anterior and a posterior cavity. The cross-sectional area of the pharyngeal region in man can be constricted while the front of the mouth is open as in /a/. A large cross-sectional area can also be produced in the pharyngeal region with either a constricted anterior passage as in /i/ or a large cavity as in /u/. The nonhuman primates cannot produce vocal-tract area functions like man's because both the apes and monkeys lack a pharyngeal region like man's (*1*, *10*), where the body of the tongue forms a movable anterior wall. We have reproduced an illustration (Fig. 4) from Negus (*10*), indicating relative positions of the palate and larynx in the nonhuman primates and in man. The nonhuman primates lack a pharyngeal region like man's, where the cross-sectional area continually changes during speech. The inability of apes to mimic human speech (*2*) is thus an inherent limitation of their vocal mechanisms. Some of man's recent ancestors also may have been unable to produce the full range of human speech; the skeletal evidence of human evolution shows a series of changes from the primate vocal tract that may have been, in part, necessary for the generation of speech (*1*). The human speech-output mechanism thus should be viewed as part of man's species-specific linguistic endowment.

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9. Some of the ape and monkey cries were apparently produced while the animals' larynges were open wide (*1*). These cries are not plotted in Fig 2 because the vocal-tract boundary conditions do not correspond to the computer model. However the acoustic analysis indicated that the shape of the animals' supralaryngeal vocal tract when they produced these cries still appeared to approximate a uniform tube. These cries therefore would not change our conclusions concerning the range of supralaryngeal vocal-tract configurations that underlie these animals' vocalizations.
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Chromosomal Location of MN Blood Group Locus

German *et al.* (*1*) have reported that an abnormal child with a translocation of a distal segment of the long arm of chromosome No. 2 to a point near the distal end of the long arm of chromosome No. 4 is hemizygous at the MN locus. They concluded that the evidence suggests that the MN locus is either in the middle of the long arm of chromosome No. 2 or near the distal end of the long arm of chromosome No. 4. In a study of the possibility of genetic linkage between a pericentric inversion on chromosome No. 2 and a number of genetic loci, Weitkamp *et al.* (*2*) reported a probability of free recombination between the MN locus and the inversion of 0.976. This calculation assumed an a priori probability of finding linkage between the MN locus and the inversion of 1 in 22, the number of autosomal pairs in man. However, if the MN locus is on either chromosome No. 2 or No. 4, then the probability that the MN locus is on chromosome No. 2 is $\Lambda/(\Lambda+1)$, where Λ, the average probability ratio, was found to be 0.51. Thus, the probability that the MN locus is on chro-

mosome No. 2 would be 33 percent and, by subtraction, that it is on No. 4, 67 percent. A further calculation involving assumptions about the genetic distance between the break points of the inversion and the translocation on the long arm of the No. 2 chromosome does not appear to refine this estimate. The data suggest that the most probable location of the MN locus is on the distal segment of the long arm of chromosome No. 4, in a region which was perhaps deleted in the child reported by German *et al.*

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Photoreception in Limulus: Role of an Electrogenic Sodium Pump?

Smith *et al.* (*1, 2*) reported electrophysiological observations on the photoreceptor cells in the ventral eye of *Limulus*. They maintain that their results cannot be explained by the conventional sodium permeability increase mechanism, but that a sodium pump with varying electrogenicity must be invoked. However, they do not appear to have taken into account the very much higher ratio of surface to volume of the *Limulus* rhabdome (*3*), as compared to that of the nerve axon. Whereas the internal ion concentrations in the axon change very little after an impulse (*4*), stimulation of a photoreceptor cell can cause very substantial changes in ion concentrations in the photoreceptor elements (*5*).

The normal resting potential (− 60 mv) and the peak receptor potential (+ 30 mv) in the *Limulus* photoreceptors (*1, 6*) can be readily derived from the Goldman equation

$$E = -\frac{RT}{F} \ln \frac{[K_i] + a[Na_i] + b[Cl_o]}{[K_o] + a[Na_o] + b[Cl_i]}$$

By substituting Adams and Hagins' values for the internal and external ion concentrations in squid photoreceptors (*7*) and values of $a = 24 \times 10^{-4}$ and $b = 0.14$ for the resting squid axon (*8*), we calculate − 65 mv for the resting potential. If we assume a sodium permeability increase upon light stimulation, and take $a = 10$ (*4*, p. 42) then a value of + 30 mv is obtained for the receptor potential (RP). The ion concentration changes, resulting from the large surface area of the membrane, would then preclude total recovery, and in the light the potential would remain about zero (*1, 6*). The essential role of the sodium permeability increase is supported by the fact that the RP is abolished by tetrodotoxin (*9*) and by the removal of sodium ions from the bathing solution (*1, 2*). As might be expected from the Goldman equation, the absence of external chloride ions has little effect on the resting potential or the RP (*1*).

Cooling the preparation, removing potassium ions from the bathing solution, or adding ouabain or calcium have essentially similar effects; they all result in an inhibition of the sodium pump, and in photoreceptors this inhibition would lead to a rapid change in ion concentrations and a depolarization of the membrane potential. Axon studies have shown that a depolarization also leads to an increased conductance (*4*, p. 64) thereby explaining why ". . . those procedures which reduce or abolish the pump adenosine triphosphatase activity correspondingly reduce or abolish the RP and affect the current and voltage (I–V) curves similarly to light" (*2*). Because of the concentration changes, the decrease in resting potential with temperature will be larger than expected from the temperature factor in the Nernst equation.

Finally, the observed net efflux of K^+ upon illumination of photoreceptor cells (*5, 10*) is difficult to reconclude with a sodium pump of constant activity but decreasing electrogenicity (*2*). Therefore, we conclude that the permeability increase theory is strengthened rather than weakened by the observations of Smith *et al.* (*1, 2*). The molecular mechanisms which might explain such a permeability increase have been discussed (*10*).

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Duncan and Bonting (*1*) do not consider those observations which are fundamental evidence against the permeability increase theory or, as we called it, the conductance increase mechanism (CIM). For example, they fail to show how our finding that, at any given level of steady-state membrane potential in the physiological range, the membrane conductance was the same or less in the light than in the dark is consistent with their theory. We interpreted our results to mean that there was no primary conductance increase with light and that the conductance increase observed was secondary and ascribable to the membrane's nonlinear current-voltage characteristic (*2*). Thus, we see the theory envisaged by Duncan and Bonting as inconsistent with these and other data reported in our papers (*2, 3*).

Duncan and Bonting suggest that the relatively high ratio of surface to volume of *Limulus* photoreceptor cells plays an important role in receptor potential (RP) mechanisms in that light leads to large changes in the intracellular ionic concentration of photoreceptors. Their evidence (*4*), however, involved a radioisotopic study of whole frog retinas, and, therefore, any ionic concentration changes observed cannot be referred specifically to the effect of light on photoreceptors alone but would also include changes in other retinal cells. The preceding remark also applies to Duncan and Bonting's subsequent comment on potassium efflux.

Duncan and Bonting cite Benolken's (*5*) and our (*2*) work as evidence that, in the light, the membrane potential remains near zero. Neither Benolken (*5*) nor we (*2*) have reported that steady light completely depolarizes a *Limulus* photoreceptor. In the steady-state the membrane potential is always inside negative (*2, 5*). Also, no one has ever reported the abolition of an RP in a *Limulus* photoreceptor with tetrodotoxin. Benolken and Russell indicated that tetrodotoxin reduced only the transient component of the RP, but required concentration a 100-fold greater than those sufficient to block all-or-none action potentials (*6*). Others, how-