The phase which precedes tonic flexion appears to be weak extension; however, there was little extensor tone. and the limbs could be manually flexed. That this phase represents a decrease in normal flexor tone is demonstrated by the fact that the animal lost its grasp if the shock was applied while it was hanging on a bar.

In seizures elicited with submaximal current, or in seizures elicited with supramaximal current during the period of postictal depression, tonic flexion was absent or greatly reduced. Thus, decrease in flexion in the sloth parallels the decrease in tonic extension seen in other animals under such circumstances (1)

Intracardiac injection of strychnine (1 mg/kg) rapidly produced a pure tonic flexor convulsion (Fig. 1, 2a). This seizure is in contrast to the strychnine convulsions in most animals in which pure extension is seen. In a more fundamental sense, however, the strychnine seizure of the sloth is comparable to that seen in other animals in that the pattern of the seizure is determined solely by the most powerful muscles. Administration of pentobarbital (35 mg/kg) produced relaxation from tonic flexion and caused the appearance of flexor jerks.

Four days after the strychnine convulsion, the spinal cord was transected at the atlanto-occipital junction, and the cord was electrically stimulated by means of a needle inserted in the cord from about C_1 to C_4 . The technique employed has been described previously (3). Spinal cord stimulation duplicated all the motor patterns seen during electroshock convulsions in the intact sloth. Single stimuli produced flexor thrusts. Stimulation at frequencies of 2 to 6 pulses per second resulted in alternating hindlimb movements which resembled the normal (upside-down) mode of locomotion of the sloth. Walking movements also are seen in the spinal cat over this frequency range (3). High-frequency stimulation (100 to 300 pulses per second) produced convulsions which contained all the components of the seizure produced by supramaximal brain stimulation. At a frequency of 300 pulses per second the onset of the flexor phase was at 4 seconds in sloth 2, only slightly less than the time to onset of flexion in the maximal electroshock seizure in this animal.

After spinal cord section in sloth 2, and just prior to cord stimulation, various reflexes of the hindlimbs were examined. Tapping the superficial tendons, the majority of which subserve flexor muscles, elicited brisk stretch reflexes. The most unusual reflex observed in this spinal sloth was that associated with painful stimulation of the

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footpad. In species previously investigated (7), this reflex consists of withdrawal of the limb to which the painful stimulus is applied and extension of the opposite limb. In the sloth the response to pinching of the footpad is strong flexion of all joints in the contralateral limb (Fig. 1, 2c). Slight flexion at the knee and marked extension of the foot and claws are observed on the side stimulated. While this reflex is opposite in direction to that which has been observed in other animals, it would appear to serve the same functions during normal locomotion, namely, to avoid the painful stimulus with one limb and to support the body with the opposite limb (8).

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References and Notes

- J. E. P. Toman, E. A. Swinyard, L. S. Goodman, J. Neurophysiol. 9, 231 (1946).
 D. W. Esplin and R. J. Laffan, Arch. intern. pharmacodynamie 113, 189 (1957).
 D. W. Esplin, Arch. Neurol. Chicago 1, Chicago
- D. W. Esplin, Arch. Neurol. Chicago 1, 485 (1959); D. W. Esplin and J. W. Freston, J. Pharmacol. Exptl. Therap. 130, 68 (1960).
 C. P. Richter and L. H. Bartemeier, Brain 49, 207 (1926).
- 4. C.
- 207 (1926).
 The animals were obtained from Ray Singleton, collector and distributor of wild animals, Rattlesnake, Fla.
 L. A. Woodbury and V. D. Davenport, Arch.

- L. A. Woodbury and V. D. Davenport, Arch. intern. pharmacodynamie 92, 97 (1952).
 C. C. Hunt and E. R. Perl, Physiol. Revs. 40, 538 (1960).
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Haptoglobin Types in Poland

Abstract. The frequency of the Hp^1 gene in Polish subjects is 0.36. This frequency is lower than that in Western European populations and higher than that in Asiatic populations. We suggest that the increase in frequency of this gene from East to West has a regular continuous character that may be attributed to a stillunclear genetic mechanism.

Distribution in different populations of the haptoglobin types and the genes controlling their inheritance is of great anthropological interest. The frequency of the Hp^1 gene is high among African Negroes and low among Asiatic people; in European populations so far tested it ranges from 0.34 to 0.47 (the reported data are collected in the paper of Sutton et al., 1). Up to now no data have been available for any of the Slavonic populations. The present report gives the results of haptoglobin type determinations of 208 Polish subjects (unTable 1. Haptoglobin types in 208 Polish subiects.

Hapto- globin type	Sex (No.)		Total
	М	F	(No.)
1-1	21	2	23
2-1	75	29	104
2–2	55	26	81
Total	151	57	208

selected blood donors from Warsaw).

Sera containing 300 mg of hemoglobin per 100 ml were assayed by vertical starch gel electrophoresis (2) with subsequent staining of gels with o-tolidine (3). The results obtained are shown in Table 1. Ahaptoglobinemia was not found in any case. The Hp^{1} frequency computed from these data was 0.3606.

The Polish population is characterized by a relatively low frequency of Hp^1 allele which is lower than that found in Western European populations (1) and close to those occurring in Norwegian (4) and Finnish (5) populations; the distribution of all three haptoglobin types in these populations is statistically indistinguishable (as proved with the chi-square test). However, haptoglobin type 1-1 occurs in 11.1 percent of Poles, 13.2 percent of Norwegians, and 14.5 percent of Finns; haptoglobin type 2-2 occurs in 38.9, 40.6, and 42.0 percent, respectively. This might correspond to a regular lowering of the Hp^1 gene frequency from the west to the east of Europewhere the only exceptions are found in some parts of Italy (6) and Germany (7)—and might be due to a historically earlier mixing of haptoglobin genes in the Polish population or to other genetic mechanisms. More detailed study of the distribution of haptoglobin types is needed to clear up this problem.

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References and Notes

- 1. H. E. Sutton, G. A. Matson, A. R. Robinson, R. W. Koucky, Am. J. Human Genet. 12, 338 (1960).
- 2. O. Smithies, Biochem. J. 71, 585 (1959).
- J. A. Owen, H. J. Silberman, C. Got, Nature 182, 1373 (1958).
- E. A. Fleischer and J. Lundevall, Proc. Sixth Congr. Hematol. (Karger, Basel, 1958), p. 906.
- 906.
 5. O. Mäkelä, A. W. Eriksson, R. Lehtovaara, Acta Genet. et Statist. Med. 9, 149 (1959).
 6. H. Harris, E. B. Robson, M. Siniscalco, in Symposium on Biochemistry of Human Ge-netics, G. E. W. Wolstenholme and C. M. O'Connor, Eds. (Churchill, London, 1960), p. 151 р. 151. 7. Н. Во
- H. Baitsch, G. Meier, L. Schoeller, D. M. Kahlich-Koenner, *Nature* 186, 976 (1960); O. Serfas and G. Schubert, *Blut* 6, 304 (1960).

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