clear that they were not diagnosing by the criteria later described by me. Nowhere in my paper is keratinization mentioned as an essential diagnostic criterion. Rather, certain definite changes of the conjunctiva were shown to be characteristic of avitaminosis A. Certainly then the biomicroscopic examinations reported in Callison's papers do not warrant the conclusions that night blindness was present in her subjects without conjunctival changes and that the biomicroscopic method is not a reliable method of detecting mild avitaminosis A.

Miss Callison raises the issue whether applying a method during the production of an uncomplicated experimental avitaminosis or during the specific treatment of the naturally occurring avitaminosis is the better test of its reliability. Either is acceptable, particularly with the support of collateral evidence. But beyond this, if a method is to be used routinely in detecting mild cases of avitaminosis, it must be applicable to the diversified natural conditions encountered in surveys of population groups.

In three surveys, routine examination for dysadaptation has failed completely to detect mild or early avitaminosis A. The data on the prevalence of avitaminosis A yielded by the method are entirely at variance with the results from diet studies, published by the Bureau of Home Economics. Furthermore, many cases of seeming dysadaptation have been found to have no basis in avitaminosis A.

In contrast, in more than one hundred individuals showing no impaired adaptation, gross or microscopic changes were present in the conjunctivae and slowly disappeared under administration of vitamin A. This evidence supports the conclusions that under ordinary conditions conjunctival changes probably precede dysadaptation in avitaminosis A and that examination of the conjunctivae is a reliable method of detecting avitaminosis A.

H. D. KRUSE

MILBANK MEMORIAL FUND

RUBBER ANALYSIS OF PLANTS IN SOUTH CAROLINA

IN 1930 and 1931 some plants growing in the vicinity of Clemson, South Carolina, were collected and analyzed for the presence of rubber. The plants were collected and identified by Mr. M. A. Rice, the chemical tests being made by the other two authors. The results are presented below. The figures given at the right show the percentage of rubber in each case. All determinations were made on a basis of air-dry weight.

Name of Plant	Per cent.
Ambrosia trifida L. Great ragweed	0.27
Amsonia Tabernaemontana Walt. Amsonia	0.26
Apocynum cannabinum L. Indian hemp	
No. 9: leaves	1.16
" 17:	0.73
Asclepias tuberosa L. Butterfly weed	
No. 5: stems	0.34
leaves	2.20
" 18:	2.21
" 22:	1.80
Asclepias sp. Milkweed	
No. 7: stems	0.38
leaves	1.93
" 10:	1.55
Asimina triloba Dunal. Papaw	0.54
Aster sp. Wild aster	1.03
Broussonetia papyrifera (L.) Vent. Paper mulberry	0.60
Cacalia atriplicifolia L. Pale Indian plantain	1.75
Decumaria barbara L. American decumaria	0.08
Erigeron annuus (L.) Pers. White-top	
Stems	0.09
Leaves	0.69
Eupatorium purpureum L. Joe-pye weed	0.45
Euphorbia corollata L. Flowering spurge	
Stems	0.26
Leaves	0.53
Euphorbia nutans Lag. Spotted spurge	0.26
Euphorbia sp. Spurge	0.70
Helianthus atrorubens L. Purple-disk sunflower	0.68
Lactuca sagittifolia Ell. Arrow-leaved lettuce	0.20
Lactuca Scariola L. Prickly lettuce	
No. 44	0.43
" 45	0.34
Lonicera japonica Thunb. Japanese honeysuckle	0.50
Morus rubra L. Red mulberry	1.02
Oxydendrum arboreum (L.) DC. Sourwood	0.24
Parthenium integrifolium L. American fever-few	1.05
Passiflora incarnata L. Maypop	0.25
Pyrrhopappus carolinianus (Walt.) DC. False dandelion	
No. 1: whole plant	0.51
" 2: stems	0.45
leaves	0.60
roots	0.61
Rhus glabra L. Smooth sumach	
Stems	0.38
Leaves	0.30
Robinia hispida L. Rose acacia	
Stems	0.29
Leaves	0.44
Sambucus canadensis L. Common elder	1.27
Silphium compositum Michx. Rosin-weed	0.74
Smilax sp. Greenbrier. (Material from a correspondent)	
No. 39: flesh of berries	1.85
" 41: flesh of berries	3.65
" 42: flesh of berries	2.05
Solidago spp. Goldenrod	
No. 3: stems	0.12
leaves	0.69
" 12: whole plant	0.89

" 23:	1.33
" 24:	1.45
Tephrosia virginiana (L.) Pers. Cat-gut	0.23
Verbesina occidentalis (L.) Walt. Small yellow crownbeard	1.22
Vinca minor L. Periwinkle	0.97
Vincetoxicum carolinense (Jacq.) Britton. Vincetoxicum	2.09

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OCCURRENCE OF VITAMINS IN FUNGI

IN reviewing the occurrence of vitamin B_2—riboflavin—Stark and others[1] mentioned that Wellstadt extracted riboflavin from *Lactarius deliciosus*, a common edible mushroom in 1935.

In 1940 a paper was published by the Academy of Science of the U.S.S.R. by G. S. Kitavin,[2] who reported that he obtained riboflavin by poisoning four-day cultures of *Aspergillus niger* grown on standard liquid media. He postulated that the mercury bichloride used as a poison resulted in physiological disturbance in the fungus which caused it to synthesize riboflavin. The riboflavin was adsorbed on a lead sulfide precipitate from which it was removed by washing with hot water. This eluant was concentrated and the solution was compared with standard solutions of riboflavin for color and fluorescence under the ultra-violet light. Crystalline riboflavin was extracted from his solutions.

Following this technique, the writers found that not only riboflavin—vitamin B_2—is produced by *Aspergillus niger*, but also thiamin—vitamin B_1. In addition to color and fluorescence tests, we used a Coleman photofluorometer which gives quantitative as well as qualitative tests. We have found in addition to *Aspergillus niger*, that other species of the higher fungi also produce thiamin and riboflavin, among which are the common market mushroom, *Agaricus campestrus, Pezzia badia,* a fleshy Ascomycete, certain species of the Glaucus group of Aspergillus, certain species of Penicillium and some of the Fusaria.

We also find that it is not necessary to add mercuric salts or other poisons to our cultures to produce vitamins, and we are able to obtain definite tests from both fungus mats or felts and media in which the fungi grow from cultures not poisoned.

From investigations carried on to date from diverse classes of fungi, it is quite evident that the production of thiamin, B_1, and riboflavin, B_2, and we suspect others, is a normal function of that group of plants known as fungi.

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[1] I. E. Stark, E. S. Gordon and W. B. Christensen, "Respiratory Enzymes," by the University of Wisconsin Biochemists, 1939, chapter 4, pp. 105.

[2] G. S. Kitavin, Plant Physiology Laboratory, Leningrad University, Academy of Science U.S.S.R., 1940, Vol. XXVIII, No. 6.

RELEASE OF POTASSIUM BY THE BRAIN OF THE DOG DURING ELECTRICAL STIMULATION

THE application of electrical tetanizing stimulus in the nerves of the leg of the *Limulus polyphemus* produces a liberation of potassium according to Cowan[1] and Young.[2] Vogt[3] has shown that a long excitation of the preganglionic cervical fibers of the dog produces a reduction of potassium in the corresponding ganglions.

In experimental epilepsy, obtained by electrical excitation of the dog's brain, Zagami[4] has found an increase of the plasma potassium and in his clinical observation, Mac Quarrie[5] has reached the same conclusion. But in both cases there are generalized muscular contractions which are probably the determinative cause of the increase of potassium. Ernst and Scheffer[6] have shown this liberation of potassium in the voluntary muscles and Cicardo[7] in the involuntary muscles.

Considering that the liberation of the potassium ions might have some relation or may be the determinative cause of the negative electropotential which originates in any tissue in activity, we assumed that a similar liberation of potassium might take place in the brain during its excitation with electrical stimulation.

In our experiments, electrical excitation of the brain of spinal or curarized dogs, in which there are no general contractions, produces an increase of potassium in the blood of the superior longitudinal venous sinus. This increase of plasma potassium is not accompanied by a similar increase of potassium in the blood simultaneously drawn from the femoral artery; which allows us to establish the cerebral origin of the liberated potassium.

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[1] S. L. Cowan, *Proc. Roy. Soc.*, B 115: 216, 1934.
[2] A. C. Young, *Jour. Neurophysiol.*, 1: 4, 1938.
[3] M. Vogt, *Jour. Physiol.*, 86: 258, 1936.
[4] V. Zagami, *Arch. Sc. Biol.*, 11: 301, 1928.
[5] Mac Quarrie, *Jour. Ann. Int. Med.*, 6: 497, 1932.
[6] E. Ernst and L. Scheffer, *Pflügers. Arch. ges. Physiol.*, 220: 655, 1928.
[7] V. H. Cicardo, *Rev. Soc. Arg. Biol.*, 17: 81, 1941.