from the ground-level $^2\Sigma$ to a hitherto unknown activated state in MgH.

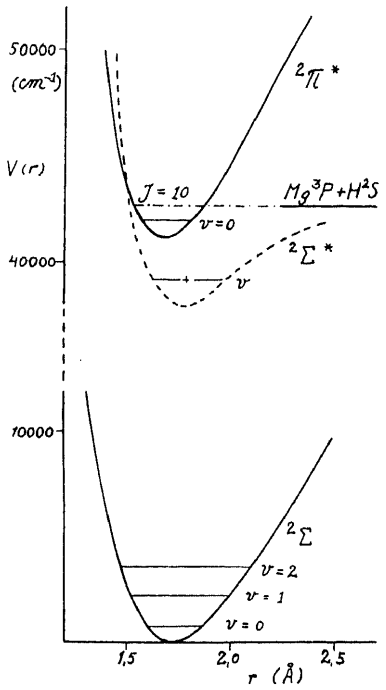

FIG. 1.

The new band consists of only R- and P-branches. Contrary to Pearse's band, this band is degraded to the red, and the R-branch forms a head at λ 2590. The last P-lines are broadened and show frequency disturbances. By means of the combination differences $R(J-1) - P(J+1)$, it can be shown that the band must be related to the ground-level $^2\Sigma$ as mentioned above. The upper state is a $^2\Sigma^*$ state, with the following constants:

$\nu_0 = 38{,}495$ cm.$^{-1}$, $B_v = 5{\cdot}44$, $r_v = 1{\cdot}79$ and $D_v = -2{\cdot}58 \times 10^{-5}$.

Kronig has predicted that the predissociation of $^2\Pi^*$ must be caused by a $^2\Sigma$-term, and it is now suggested that this term is identical with the new $^2\Sigma^*$-level. This suggestion is illustrated in Fig. 1, where the potential curves of $^2\Sigma$, $^2\Sigma^*$ and $^2\Pi^*$ are drawn. It is very probable that $^2\Sigma^*$, in conformity to the D, $^2\Sigma$-term of CaH, dissociates into a normal H and an activated ^3P metal atom, which is supposed to have the same energy as the predissociation limit at $J = 10$, $v = 0$ of $^2\Pi^*$. The essential transition between $^2\Sigma^*$ and $2\Pi^*$ will then, in this case (MgH), take place at the interior part of the potential curves, where they are in contact with each other.

In addition to the R- and P-lines, which are present in the emission spectrum of Pearse's band, some new lines are recorded in absorption, which are related to higher rotational terms in $^2\Pi^*$. These lines are also somewhat diffuse.

A complete description of these phenomena will be published shortly.

B. GRUNDSTRÖM.

Laboratory of Physics,
University, Stockholm.
Nov. 28.

[1] *Proc. Roy. Soc.*, *A*, **122**, 422; 1928.

A Method of Determining the State of Degeneration of a Gas

VARIOUS investigators have tried[1] to find an experimental method by which to determine if gases like hydrogen and helium are degenerated or not at very low temperatures[2]. All attempts of this kind have hitherto been unsuccessful. The principal difficulty results from the fact that the technique of the experimental methods used (measurements of the specific heats or the velocity of sound) do not permit of measurements at very low pressures where the van der Waals' forces have no influence.

We believe we have found an experimental method which will give without ambiguity a solution of the problem of degeneracy of these gases. This method is partially based on the following idea, which was put forward so long ago as 1881 by Stokes[3]. In the classical kinetic theory of gases, the following formula has been established for the coefficient of viscosity η: $\eta = \frac{1}{3}\rho\bar{c}.l_0$, where ρ is the density, $\bar{c}$ the mean velocity and l_0 the mean free path. As l_0 is proportional to ρ^{-1}, it follows that in the classical theory η is independent of pressure. But when the dimensions of the apparatus are such that the mean free path of the gas molecules does not exceed a certain value, η must become a linear function of the density. It should also be noted that from the direction of this straight line, $\bar{c}$ can be calculated without difficulty.

I made such experiments with one of my students, Mrs. A. Claes. These measurements will be published shortly in more detail. The viscosity of oxygen gas is measured at 293°, 90° and 72° K. From these experiments, the ratio of the direction coefficients of the three straight lines are calculated and it is found that the ratios correspond respectively within a few per cent with $(293/90)^{1/2}$ and $(90/72)^{1/2}$. This is in agreement with the classical value of $\bar{c} = 2(2kT/\pi m)^{1/2}$.

In order to detect if degeneracy occurs at very low temperatures, preparations are being made for similar measurements with helium gas at liquid hydrogen and at liquid helium temperatures; and an attempt will also be made to make the experiments more accurate. The ratio found between the mean velocities will enable us to conclude whether degeneracy exists or not.

A. VAN ITTERBEEK.

Physical Laboratory,
Louvain.
Dec. 16.

[1] W. Meissner, *Phys. Z.*, **29**, 897 (1928). W. H. Keesom and A. Van Itterbeek, *Proc. Amst.*, **34**, 996 (1931), Comm. Leiden No. 276*d*. M. Sôto, *Tôhoku Univ. Sci. and Tech. Reports*, **24**, 26 (1935).
[2] A. Einstein, *Berl. Ber.*, 261 (1924); 3 and 18 (1925). E. Fermi, *Atti Lincei* (6), **3**, 145 (1926). *Z. Phys.*, **36**, 902 (1926).
[3] G. G. Stokes, *Phil. Trans.*, **2**, 435 (1881).

Interrelationship of Vitamins

THE successful study of vitamins depends to a large extent upon the production of uncomplicated symptoms in experimental animals which can be cured by purified preparations of the vitamin in question. However, the cure of certain symptoms even with crystalline preparations does not necessarily mean that the deficiency was due to a lack of the factor fed. We have recently encountered such conditions in our laboratory and wish to describe one such relationship briefly.

Typical symptoms due to vitamin B_4 deficiency in rats have been described by Reader[1], but many workers have experienced considerable difficulty in

producing these symptoms. This is undoubtedly due to the fact that a highly purified synthetic diet is used which may be low in constituents of the vitamin B complex not yet recognised. Deficiencies other than vitamins B_1, B_2, and B_4 in purified chick rations are readily evident. When using diets known to be low in vitamin B_1 for rats, characteristic vitamin B_4 deficiencies occurred. The results were most striking in the case of our ration 240 A[2], which is an autoclaved natural grain ration. If rats are placed on this ration on weaning they do not show polyneuritis within two weeks, which is so characteristic of chicks reared on the same diet, but they fail to grow after two weeks and remain very constant in weight for a number of weeks. When they have been on the ration four to six weeks, typical B_4 avitaminosis occurs. The back is hunched, their jowls protrude, they walk high on their rear legs and show a wobbly gait. In some rats typical polyneuritis may appear two to four weeks later. Few workers seem to have recognised the distinctly different symptoms in the two types of deficiency. Guerrant and Dutcher[3] recognise different symptoms in the rats which succumb early and those that survive longer.

If the rats showing vitamin B_4 deficiency symptoms are given vitamin B_1 (Ohdake concentrate) the symptoms gradually disappear, but the animals do not show the dramatic response obtained in animals with uncomplicated polyneuritis. This fact might lead to the conclusion that vitamin B_1 also cures paralysis in rats. However, we believe that another explanation is more plausible. The autoclaved ration produces a chronic vitamin B_1 deficiency including a rather severe anorexia, but the animal makes a terrific struggle to counteract polyneuritis. This is done partly by reducing its food intake, since this will reduce the accumulation of carbohydrate intermediate products, and partly by consumption of fæces. It is practically impossible to prevent coprophagy. According to present ideas, the yeast[4] in the digestive tract would supply some vitamin B_1 but very little vitamin B_4. The basal ration contains some vitamin B_4, but the decreased food consumption reduces the intake of this factor below the minimum requirement and vitamin B_4 deficiency results. When vitamin B_1 is fed, it does not cure the paralysis, but relieves the anorexia, and food consumption increases to a level which supplies sufficient vitamin B_4.

We feel that these facts introduce some interesting questions in vitamin studies. First, synthetic diets used for vitamin B_1 studies must be amply supplied with vitamin B_4, especially if only growth is used as the criterion of potency. Secondly, the improved growth obtained by certain workers by adding large amounts of B_1 to rats on a B_1 low diet may be due to the additional effect of traces of B_4 present as an impurity in the B_1 preparations. Thirdly, greater attention must be given to the interrelationship of vitamins both in the laboratory and in clinical practice.

C. A. ELVEHJEM.
AARON ARNOLD.

Department of Agricultural Chemistry,
University of Wisconsin,
Madison, Wisconsin.

[1] Reader, V., *Biochem. J.*, **24**, 1827 (1930).
[2] Kline, O. L., Keenan, J. A., Elvehjem, C. A., Hart, E. B., *J. Biol. Chem.*, **99**, 295 (1932).
[3] Guerrant, N. B., and Dutcher, R. A., *J. Nutr.*, **8**, 397 (1934).
[4] Guerrant, N. B., Dutcher, R. A., and Tomey, L. F., *J. Biol. Chem.*, **110**, 233 (1935).

Decomposition of Adrenalin in Tissues

PHARMACOLOGICAL evidence suggests that adrenalin is actively destroyed in tissues. We have studied the effect of tissue on decomposition and loss of pharmacological activity of adrenalin.

Tissue slices in Ringer solution or extracts of tissue were incubated in manometer flasks with adrenalin, and after suitable times the adrenalin activity of the fluids was tested on rabbit's gut. Rat liver slices in Ringer solution (phosphate buffered, *p*H 7·3) incubated with adrenalin accelerate the loss of adrenalin activity as compared with controls without tissue. This decomposition of adrenalin occurs only in the presence of oxygen. Whereas the oxygen uptake of liver slices is not noticeably influenced by adrenalin, the oxygen uptake of rat liver extracts is considerably increased when adrenalin is added.

Comparison of the extra oxygen uptake under these conditions with the oxygen consumption of adrenalin in Ringer solution alone shows the following difference: in the last case the adrenalin activity is approximately halved after the uptake of 2–3 atoms of oxygen per molecule of adrenalin; in the presence of tissue extracts the activity is about halved after the uptake of one, and practically abolished after the uptake of two atoms of oxygen per molecule.

The significance of these findings and their relations to the work of others will be discussed elsewhere.

H. BLASCHKO.
H. SCHLOSSMANN.

Physiological Laboratory,
Cambridge.
Dec. 17.

Contamination in Petri Dish Boxes

IN the course of investigations carried out on the influence of bacteria and fungi on flour kept under storage conditions, considerable difficulties were encountered by repeated contamination of the Petri dishes used for spore counts by 'spreaders' such as *Bacillus dendroides*. Fig. 1 shows an example of the type of contamination that was obtained. The presence of these 'spreaders' necessarily vitiates counts on such contaminated plates.

FIG. 1.

Tests were made to discover the cause of this contamination and at which point it occurred, and it was eventually found that the metal boxes used for sterilising Petri dishes were at fault. The atmosphere in the St. Albans laboratories is heavily contaminated with bacteria and fungi owing to the