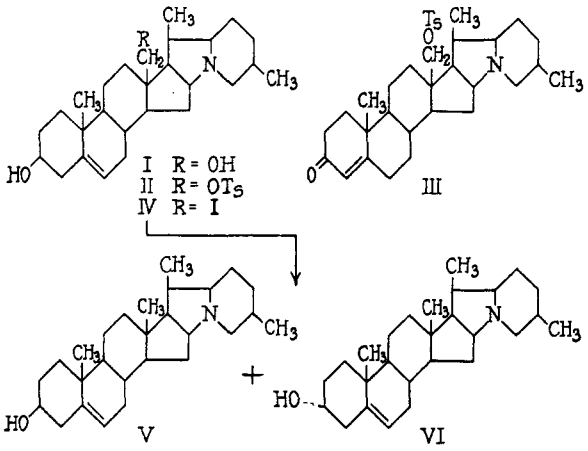
ketone gave an almost quantitative yield of (IV), m.p. 294–297° dec.; $[\alpha]^{30}D$ −38° (*c* 1.0 in abs. EtOH); *Anal.* Calcd. for $C_{27}H_{42}INO$: C, 61.94; H, 8.09; I, 24.24. Found: C, 62.00; H, 8.15; I, 24.12.

Attempts to replace the iodine of IV with hydrogen by treatment with a zinc–copper couple, aluminum amalgam, hydrogen and palladium on calcium carbonate, or with zinc and acetic acid were not successful. However, reduction of IV with sodium in ethanol furnished a good yield of a mixture, m.p. 185–235° dec., which was separated with digitonin into two pure compounds. The first of these (V) was identified as solanidine (3(β)-hydroxy-Δ^5-solanidene), m.p. and mixed m.p. with authentic solanidine, 216–218.5°; $[\alpha]^{34}D$ −27.1° (*c* 0.54 in chf.); *Anal.* Calcd. for $C_{27}H_{43}NO$: C, 81.54; H, 10.91. Found: C, 81.27; H, 10.95. The infrared spectrum of V proved to be identical in all respects with that of authentic solanidine. The second component, m.p. 238–239°, $[\alpha]^{34}D$ −12° (*c* 1.5 in chf.); *Anal.* C, 81.43; H, 11.12, is isomeric with solanidine. Because of its derivation from IV, the similarity of its infrared spectrum to that of solanidine, and its behavior toward digitonin, it is believed to be 3(α)-hydroxy-Δ^5-solanidene (VI). It results presumably from an epimerization accompanying the sodium reduction. Confirmatory work on the structure of VI is in progress.



While the transformation of isorubijervine to solanidine leaves no doubt that isorubijervine is a hydroxy-solanidine, the position of the primary hydroxyl group remains to be settled. The strongly hindered character of the primary iodide group of IV toward the reducing agents cited is in accord with the previous arguments for assigning the hydroxyl group to the 18-position.[7] On the basis of these considerations, irorubijervine is assigned the structure of Δ^5-solanidene-3(β),18-diol (I) and the intermediates the structures represented by II and IV.

All analytical data have been obtained by Mr. D. Rigakos of this laboratory.

THE ROCKEFELLER INSTITUTE
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NEW YORK 21, N. Y.

S. W. PELLETIER
WALTER A. JACOBS

RECEIVED JULY 17, 1952

THE NATURE OF THE B–N BOND IN B-TRICHLOROBORAZOLE, BORON NITRIDE AND BORON TRICHLORIDE

Sir:

From their recent structure determination of crystallized $B_3N_3H_3Cl_3$, Coursen and Hoard[1] have shown that the B–N bond length is 1.413 ± 0.01 Å. in this compound. By comparing this value with that found in $B_3N_3H_6$ (1.44 ± 0.02 Å.) they have suggested that double-bond resonance in the B_3N_3 ring must be at least as fully excited in $B_3N_3H_3Cl_3$ as in $B_3N_3H_6$. This implies that the predominant electron configuration must be (A) rather than (B) (Fig. 1), since (B) cannot contribute in $B_3N_3H_6$.

The result obtained for the B–N bond length in boron nitride (1.446 Å.)[2] supports both this contention, and also that of graphite-like resonance in BN, since the observed difference between the two bond lengths (0.033 ± 0.01 Å.) agrees with that expected (0.035 Å.)[3] as a consequence of the required increase in double-bond character of $B_3N_3H_3Cl_3$ as compared to BN.

However, the observed length of the B–Cl bond in $B_3N_3H_3Cl_3$, being the *same* as that observed in BCl_3 to within 0.02 Å.,[1] conflicts with this view: for in BCl_3 the bond is said to be part-double,[4] so that configuration (A) requires a longer B–Cl bond than in BCl_3 by about 0.10 Å. Configuration (B) will certainly not do: it requires not only a *shorter* B–Cl bond than in BCl_3, but also a *longer* B–N bond than in BN.

There is also the possibility of configuration (C). If it is supposed that in BN and BCl_3 there are only single bonds (which are shortened from the sum of Pauling's covalent tetrahedral radii by the deficiency of electrons round the boron), then (C) predicts the same boron radius in all three compounds.

The situation is summarized in Table I.

TABLE I

BOND LENGTHS IN $B_3N_3H_3Cl_3$

Bond	Observed	Predicated by configurations A	B	C
B—N	1.413 ± 0.01	1.411[a]	1.55[a]	1.446[b]
B—Cl	1.760 ± 0.015	1.87[a]	1.65[a]	1.76[b]

[a] Assuming 1/3 part-double-bond in BCl_3 and BN. [b] Assuming single-bond and sextet boron in BCl_3 and BN.

Clearly, neither (A) nor (B) can alone account for the bond lengths observed in $B_3N_3H_3Cl_3$. However, a combination of two-thirds (A) and one-third (B), which gives a one-third part-double character to both the B–Cl and the B–N bond, will, on the assumption of similar bonds in BN and BCl_3, give as good a fit as (C).

There is likely, however, to be some real difference between the B–N bond in BN and in $B_3N_3H_3Cl_3$ to account for the observed difference in length. This may be a difference between the contribution from (C) in the two substances. Without definite information on the bond type in BN and BCl_3, the bond lengths do not yield infor-

(1) D. L. Coursen and J. L. Hoard, THIS JOURNAL, **74**, 1742 (1952).
(2) R. S. Pease, *Acta Cryst.*, **5**, 356 (1952).
(3) L. Pauling, *Proc. Roy. Soc.* (*London*), *A* **196**, 343 (1949).
(4) L. Pauling, "The Nature of the Chemical Bond," Cornell University Press, Ithaca, N. Y., 1945.

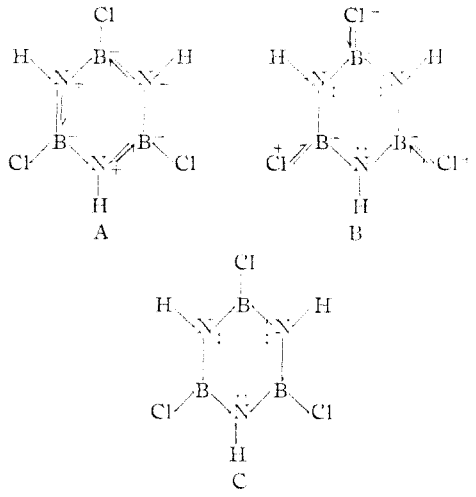

Fig. 1.

mation on the magnitude of this contribution. But the electrical properties of BN[2] and the constancy of the bond length observed in the series BF_3, $BMeF_2$, BMe_2F, BMe_3[5] suggest that the single-bond configuration might be important in some trigonally coördinated boron compounds as well as in the tri-aryls and tri-alkyls.

(5) S. H. Bauer and J. M. Hastings, THIS JOURNAL, **64**, 2686 (1942).

ATOMIC ENERGY RESEARCH ESTAB. R. S. PEASE
HARWELL, BERKS, ENGLAND

RECEIVED JUNE 2, 1952

AN ANTISEROTONIN WHICH IS ORALLY EFFECTIVE

Sir:

Recently, antimetabolites of serotonin have been described which overcome the constriction of segments of arteries elicited by this naturally occurring vasoconstrictor.[1] The suggestion has also been made that such antimetabolites might merit consideration as pharmacological agents capable of influencing favorably some kinds of constriction of vessels seen in higher animals. More recently, it has been found that intravenous administration of 2,3-dimethyl-5-aminoindole will prevent the rise in arterial blood pressure of dogs which follows the intravenous injection of serotinin.

If one envisions a desirable agent for clinical use in a condition such as hypertension, one of the first requisites is that this agent be effective by the oral route. Intravenous administration is so objectionable that the need for it would render an otherwise promising substance impractical. When the aminoindoles were given to dogs orally, and the subsequent effect of serotonin on the arterial blood pressure[2] was examined, it was seen that, although the rise in pressure could be inhibited partially, a large dose of analog was required. This was not too surprising since tissues are known to contain enzyme systems which destroy *p*-phenylenediamines, and these analogs are substituted *p*-phenylenediamines. Therefore, an analog of serotonin was sought which would be active by oral administration.

(1) D. W. Woolley and E. Shaw, THIS JOURNAL, **74**, 2948 (1952).
(2) I. H. Page, *J. Pharmacol. and Exp. Therap.*, **105**, 58 (1952).

The corresponding 5-nitroindoles were found to do this, as the following experiment will show. A normal dog was anesthetized with nembutal, and a mercury manometer was connected through a needle to the femoral artery (without surgical operation).[3] The response to intravenous serotonin[4] was noted (Fig. 1). Being thus of proven reactivity, the dog was fed daily 500 mg. of 2-methyl-3-ethyl-5-nitroindole for 4 days, and again challenged with serotonin. The figure will show that even twice the dose of the vasoconstrictor elicited no significant effect. This experiment was repeated in other dogs with similar results.

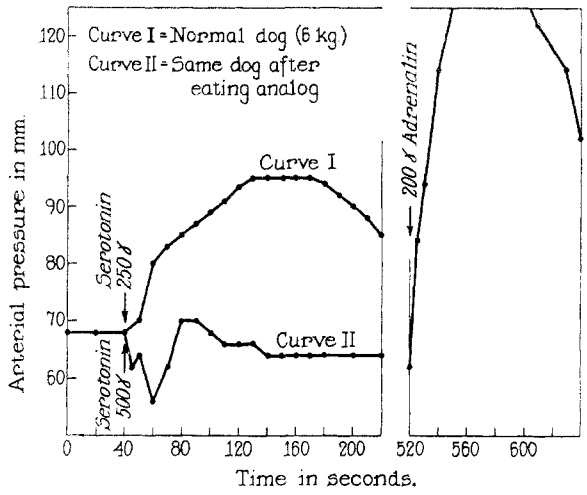

Fig. 1.

This nitroindole was inactive as an antimetabolite of serotonin when tested *in vitro* with artery rings.[1] Its activity *in vivo* suggested that the real antimetabolite, *i.e.*, the aminoindole, was transported in this protected state to the site of action, and there liberated by reduction.

The nitroanalog seemed to be relatively harmless to normal animals. No toxic manifestations have been seen in mice fed it as 1% of their ration. Similarly, dogs fed 500 mg. per day for four days showed no ill effects. Note also in the figure that the normal arterial pressure was not lowered by such feeding.

FROM THE ROCKEFELLER INSTITUTE FOR MEDICAL RESEARCH, NEW YORK D. W. WOOLLEY[5] E. SHAW

RECEIVED JUNE 26, 1952

(3) We are greatly indebted to Dr. I. L. Schwartz for skillfully carrying out these operations.
(4) Serotonin was kindly supplied by the Abbott Laboratories. The weights stated are of serotonin creatinine sulfate.
(5) With the technical assistance of G. Schaffner.

ACYLATION OF 17α-HYDROXY-20-KETOSTEROIDS: COMPOUND L DIACETATE

Sir:

Although acetylation of the C-17 hydroxyl group of 17β-hydroxy-20-ketosteroids can be effected relatively easily with hot acetic anhydride and pyridine,[1] acetylation of the epimeric 17α-hydroxy

(1) C. W. Shoppee and D. A. Prins, *Helv. Chim. Acta*, **26**, 185 (1943); J. von Euw and T. Reichstein, *ibid.*, **30**, 205 (1947).