thetic activities and are equipped to meet all emergencies by adaptation, they should not be capable of developing alternative pathways or other shunts to meet the needs. The true chemotherapeutic agent shows the specific selective action by the above-mentioned mechanism. The antiseptic, on the other hand, not possessing this selective action, when put in a complicated system, gets entangled in the one it comes across first and thus, going astray, is not available where its action is required. This is how the antiseptic which is very active in the test-tube looses its activity *in vivo*.

One of the ways to stop an enzyme system from functioning in a selectively specific way is by the mechanism of competitive inhibition, taking advantage of the structural specificity of the substrate of any other participant in the enzyme system. A compound which is close enough in structure to this to get involved in the first stage but not identical enough to be actually utilised in the enzyme reaction can stop the enzyme system from functioning. If this enzyme system is itself vital or a vital link in an important chain, we have obtained the typical chemotherapeutic effect. . If this effect is to be of clinical value, the additional conditions to be satisfied are: (*i*) the inhibitor should not undergo metabolisation or have affinity for other compounds *in vivo* and (*ii*) the substrate or compound being displaced by the inhibitor should not be produced *in vivo* in sufficient concentrations to nullify the action of the inhibitor itself. Though we are able to chalk out these principles in concrete terms, no great advance has been made in discovering more chemotherapeuticals because we do not know enough about the chemistry of the enzyme systems involved in bacterial multiplication and proliferation. Strangely enough, we came to know of the role of p-aminobenzoic acid by the reverse process of working with a true chemotherapeutic agent. Thus, the mechanism of action of the chemotherapeuticals gives us a clue to the understanding of the chemistry of bacterial multiplication.

Then there is the question as to the exact phase of bacterial growth on which the drug should act to obtain striking chemotherapeutic action—whether it should affect the respiration, metabolic or catabolic reactions, the cell division, etc. This action will decide the nature of the antibacterial effect obtained. The sulpha drugs and penicillin show their effect only when the bacteria are rapidly multiplying and not when they are in the stationary phase. Their effect is not, therefore, observed at once. This is roughly taken as a bacteriostatic action as differentiated from the bactericidal effect in which the lethal action is immediate. If an immediate chemotherapeutic effect is desired, the action of the drug must be directed against even the stationary phase of bacterial growth. We do not as yet know enough about the bacterial enzyme systems to evolve anything useful in this direction.

Physicochemical Theory of Chemotherapeutic Action

The theory of action of the sulpha drugs, by displacing *p*-aminobenzoic acid from an enzyme system by competitive inhibition, has provided a solid base on which to build the physicochemical theory of the intensity of the chemotherapeutic effect. One fortunate fact helping us in this venture is the structural simplicity of the drugs in which the only variable is the substituent at the sulphonamide radical. So the problem is to find out how this substituent governs the degree of the intrinsic therapeutic activity. Since the mechanism involved is competitive inhibition, the more the sulpha drug resembles *p*-aminobenzoic acid the greater the degree of activity. Though the *p*-aminobenzoic acid ion and the *p*-aminobenzenesulphone radical resemble each other in geometric configuration, the only distinct feature about the former is the negative charge. So the more negative the sulphone group, the greater the activity of the sulpha-radical The only way of gauging the negativity of the sulphone group (governed by the attached amino or substituted amino group) is by the acid dissociation constant (pKa) of the drug. The theoretical calculation shows that the maximum activity will be shown by that drug whose pKa value is 6·7. On this basis the maximum activity is almost reached in sulphathiazole (pKa=7·12), sulphadiazine (pKa = 6·48) and sulphamerazine (pKa = 7·06). That no drug has so far been discovered which shows greater activity than the above, indicates that the theoretical reasoning is sound. This is the first time in the history of chemotherapy that a physicochemical property of a compound could be used to predict its antibacterial activity. In the light of this, attempts are also being made to treat the problem on a physicochemical basis. As stated before, this has introduced a new outlook and logic in the field of chemotherapy which has earned for it the status of a science.

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MEDIUM OF STUDIES IN COLLEGES

IN the Dominon Parliament, on the first of March, Maulana Abul Kalam Azad, Education Minister, said in reply to Seth Govind Das that as far as primary and secondary education was concerned, the Provincial Governments had accepted the principle that the medium of instruction should be the mother-tongue. Every effort was being made to put this into practice.

The Central Advisory Board of Education and the Educational Conference both came to the conclusion that the change in University education should be by stages, so that the standard of education did not suffer. It was agreed that the change-over should be spread over five years, and in the sixth year all education should be in the Indian language or languages which should be the medium of instruction. The English language would, however, continue to be a second language and a subject for post-graduate studies.