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COMBINATIONS OF ANTIMICROBIAL DRUGS

The indiscriminate use of antimicrobial drugs has been much condemned. There are several important disadvantages and dangers. The use of combinations of these drugs introduces additional problems and risks, among which is an increase in the incidence of adverse reactions and in sensitization.

The possible indications for combined use of these drugs have been stated by Jawetz.[1] In certain patients who are extremely ill with infection of unknown cause, it might be advisable to use more than one antimicrobial drug, while every effort is being made to establish the correct diagnosis. In mixed infections a combination of drugs aimed at a complex flora may be more effective than a single drug. Infections of the skin, wounds, body cavities, and of the cardiovascular, respiratory and urinary systems, may necessitate this form of attack. In certain infections where resistant organisms develop, an additional drug may delay the emergence of resistant strains. This has been well established in the chemotherapy of tuberculosis, but it may apply also to other chronic infections. The administration of a combination of drugs may in certain instances decrease adverse reactions, since each of the administered drugs may be given in a smaller dose below the level of adverse reaction. There are occasions when one drug may enhance the action of another on a particular microorganism; one of the best examples of such 'synergism' is the use of penicillin with streptomycin in bacterial endocarditis due to *Strept. faecalis*. Desirable combinations of drugs have to be established or suggested by appropriate laboratory methods.

The mechanism involved in synergism is not properly understood at present. One drug may be more effective on one type of microorganism, and the other on the remainder of the microbial population; or one drug may block microbial replication incompletely and the second drug then produce bactericidal effects. Thus the bactericidal action of penicillin on *Streptococcus viridans* is enhanced by the presence of streptomycin. The possibility of a second drug actually diminishing the effectiveness of the drug with which it is given in combination is also to be borne in mind. Fortunately, in clinical practice this is not a frequent occurrence, but there are a few documented examples of 'antagonism'. Penicillin and chlortetracycline (Aureomycin) were found to cure fewer patients with pneumococcal meningitis than when penicillin was used alone. There are other examples of such antibiotic antagonism. The mechanism must be the result of sequential action whereby the interfering drug reduces the activity of that metabolic pathway that would have been inhibited by the bactericidal drug. An interfering agent such as tetracycline acts primarily as an inhibitor of protein synthesis and antagonizes penicillin, which acts as an inhibitor of mucopeptide synthesis by the cell wall. It may be that protein synthesis must proceed actively for active mucopeptide synthesis to occur, and so inhibitors of protein synthesis antagonize inhibitors of mucopeptide synthesis.

The results of *in vitro* tests have correlated very poorly with the clinical results;[2] a mixture of antimicrobial drugs is superior to a single agent only in a few instances. It is remarkable that there has been such readiness and enthusiasm to accept claims made for mixtures of antimicrobial drugs. Particularly condemned have been the 'fixed dose' forms. This use of drugs does not offer the physician discretion in the choice of components or of the ratios in which they are used. 'The use of such "fixed dose" antibiotic mixtures and the manner in which they are being exploited represent a major backward step in the management of infections.'[3] Inadequate treatment is encouraged because there is the tendency to use the same total dose of mixture as of a single agent. Also, a false sense of security is created when in fact a narrower rather than a wider coverage is supplied. Antibiotics should generally be selected individually, each for its own value, and administered in the proper dose for the intended purpose.

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THE RACIAL INCIDENCE OF TESTICULAR TUMOURS*

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It is generally accepted that tumours of the testis of the seminoma/teratoma type are uncommon in non-White patients. Dixon and Moore[1] state that only 1·5% of these tumours in their series occurred in non-Whites, although the population at risk contained up to 8·5% non-Whites. Kochler *et al.*,[2] in a series of 1,127 testicular tumours collected from a population which contained 50% Negroes, found only 17 cases (1·5%) in the non-White group. Cohen and Tomskey[3] found only 5 cases (8·9%) in Negroes, out of 56 tumours, and all of these were in undescended testes.

Groote Schuur Hospital, Cape Town, admits patients from 3 racial groups: White, Bantu and a group of mixed blood known as Cape Coloured. It was thought worth while to study the racial incidence of testicular tumours within this population to discover whether it followed the same trends as elsewhere.

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MATERIALS

During the 28-year period 1940 - 1967, 62 testicular tumours from patients admitted to Groote Schuur Hospital were seen in the Department of Pathology. Slides and/or blocks were available for study in all cases except one which was excluded.

Teratoma were the most frequently seen (24 cases: 39%), followed by seminomas (19 cases: 30%). Only 4 cases were examples of combined seminoma and teratoma. In addition there were 2 examples of interstitial-cell tumour, 1 Sertoli-cell tumour, 1 orchioblastoma, 7 cases regarded as primary lymphoma and 2 secondary tumours (1 from the prostate and 1 from the large bowel). The nature of one tumour was not decided.

The classification employed was that of the Testicular Tumour Panel and Registry of the Pathological Society of Great Britain and Ireland.[4] When so classified the teratoma group contained 1 differentiated teratoma, 14 malignant teratomata (intermediate type A), 7 malignant teratomata (intermediate type B), and 2 choriocarcinomas.

During the period under discussion the relative admissions for the 3 racial groups were: 47% White, 44% Coloured and 9% Bantu.

RESULTS

The majority of the seminoma/teratoma group of tumours (68%) occurred in the White group and only 32% in the non-White group (Table I). Of the remaining 13 tumours, both interstitial-cell tumours occurred in White patients, 3 of the 7 lymphomas occurred in White patients and the remainder in Coloured patients, the single orchioblastoma occurred in a Coloured child, the Sertoli-cell tumour occurred in a Bantu, and both secondary tumours occurred in White patients.

TABLE I. INCIDENCE OF TESTICULAR TUMOURS

	White		*Coloured*		*Bantu*		*Total cases*
	Cases	%	*Cases*	%	*Cases*	%	
Seminoma	13	68·5	5	26	1	5	19
Teratoma	18	75	6	25	–	–	24
Combined	1	–	3	–	–	–	4
Total	32	68	14	30	1	2	47

The age incidence of the seminoma/teratoma group corresponds with that of most other series, in that most seminomas occurred during the 4th and 5th decade, while the teratomata occurred maximally a decade earlier. There was no difference between White and non-White with regard to age incidence.

Four of the tumours occurred in undescended testes: an incidence in the same range as occurred in most other series. Of these, 2 were seminomas, 1 was a teratoma and 1 a combined tumour.

DISCUSSION

The lower incidence of testicular tumours in the non-White races is, according to the literature, restricted to the seminoma/teratoma group. The series presented was derived from a hospital population comprising 47% White and 53% non-White patients. The incidence of these tumours in the non-White group is only 32%, but is considerably higher than in other series.

Comparisons of the racial incidence of a given tumour are not valid unless the relative ages of the racial groups are taken into account. In this population the average age of the White patients is approximately 1 decade above that of the Coloured, which in turn is 1 decade above that of the Bantu. As this variation is most marked above the age of 60 years and most of the tumours in this series occurred below the age of 50 years, this difference in ages has little significance.

The majority of non-White patients in this series were Cape Coloured, and there was only 1 Bantu patient. A single case in a small series cannot be accurately converted into a percentage, but does indicate an incidence similar to that reported in the USA. This similarity may be of significance in that the American Negro has an African ancestry. The Cape Coloured, on the other hand, comprise a distinct ethnic group of European, Hottentot and Malay descent.

It has therefore been confirmed that there is a lower incidence of testicular tumours in the non-White races, but that the difference is not as marked in the Cape Coloured group as in the non-Whites of other series.

SUMMARY

The racial incidence of testicular tumours of the seminoma/teratoma type has been investigated in a hospital population comprising 47% White, 44% Coloured and 9% Bantu patients. Of 47 tumours of this type seen, 32 (68%) occurred in White patients, 14 (30%) occurred in Coloured patients and 1 occurred in a Bantu.

As is generally accepted, the incidence of these tumours in non-White patients is lower than in the White patients, but in the case of the Cape Coloured this incidence is higher than previously reported figures.

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