NEW SEA-WALL AT ABERDEEN

THE subject of coastal protection is always with us but is seldom nationally publicized until a catastrophe occurs, when a combination of storm and excessively high tides causes the sea to overwhelm existing defences, flooding land behind them, resulting in grave damage to both life and property, to say nothing of the sea-walls and groynes concerned.

Recent work on a section of the coastline between the north pier of Aberdeen harbour and the mouth of the River Don is a case in point. In this example, lack of toe piling coupled with erosion of foreshore during the winter of 1961–62 determined the complete collapse of some 100 ft. of the then existent sea-wall and caused severe damage to the front of the apron over several hundred feet. According to *Concrete Quarterly* (No. 61, April–June 1964; Cement and Concrete Association, London), "The new work now nearing completion consists of two parts. The first and minor portion consists in rebuilding and strengthening portions of the existing wall extending some 2,500 ft. north from the beach bathing station. . . . A completely new wall has been built over the remaining 5,000 ft. or so of sea-front from the existing wall to the Don. A flexible facing of precast concrete blocks, with a post-tensioned precast coping, has been employed".

The construction of flexible revetments by mortaring stone or masonry blocks with a flexible material is a technique which has been known for centuries, ". . . a revetment on the banks of the River Tigris in ancient Assyria, built in 1300 B.C. of limestone blocks and brickwork mortared with bitumen and bituminous mixtures, is still in use to-day, and in good condition, more than 3,000 years later". This new sea-wall at Aberdeen uses much the same formula, only precast concrete blocks instead of limestone, etc., with bituminous jointing material, on a slope of 1 : 2. At the foot the wall is supported by steel sheet-piling topped with an *in situ* concrete capping beam. "Precast ribs at 29-ft. 5-in. centres span between the footing at the bottom and the *in situ* concrete foundation to the precast concrete coping at the top, and give lateral rigidity to the blockwork." It is recorded that some 42,000 precast blocks were involved in this project; these were vacuum-cast on the Miller system; this permits de-moulding to be done within about 4 min; also the use of steel moulds enabled thousands of components to be cast from the same moulds.

Construction of new groynes was an integral part of the scheme, not only to arrest northward drift of littoral sand, but also to help protection of the shore; these groynes are of greenheart timber. Finally, to make doubly sure, and to counteract erosion and stop scour at the northern end of the impermeable wall, there is a terminal section of gabions—permeable wire cages filled with stone—used as a transition section between the wall itself and the existing beach. The illustrations accompanying this article in *Concrete Quarterly* adequately convey the essential constructional details of this new sea-wall, equally a sense of the solidarity of this recent addition to Britain's coastal protection, a matter of great credit to all the engineers concerned.

CHROMOSOMES AND HUMAN MALFORMATION

IN the space of a few years, the enquiry into human variability has been raised from a somewhat speculative stage to the level of exact observation, said Prof. J. A. Böök in a lecture * entitled "Some Mechanisms of Chromosome Variations and their Relation to Human Malformations". The exact observations referred to chromosomal investigations, which have been made possible in recent years through improved techniques of culturing human cells *in vitro*.

Since 1959, when it was first demonstrated that certain chromosomal abnormalities were associated with specific congenital malformations[1,2], a large number of instances have been reported. These cytological abnormalities, Prof. Böök pointed out, were not new cytological phenomena, but demonstrated that man, like other animals and plants, was occasionally subject to abnormalities in the structure of the hereditary system.

The occurrence of an extra chromosome (trisomy), arising from normal parents by chance meiotic or mitotic non-disjunction, was the commonest chromosomal abnormality. In most instances the origin of the abnormal karyotype could be correlated with above-average maternal age—thus revealing the increased risk of chromosomal accidents by ageing. In addition to the three well-established trisomic states [13–15 syndrome, 17–18 syndrome and Downs syndrome (trisomy 21)], the possible differentiation of a fourth syndrome was outlined—involving trisomy for a small acrocentric chromosome—presumably No. 22. From five presumptive cases of this 22 trisomy, the defects in common included microcephaly, deafness, large, low-set malformed ears and pre-auricular papillomata.

The presence of a complete extra set of chromosomes (triploidy) still constitutes a rare abnormality and, apart from the sporadic occurrence in spontaneously aborted fœtuses[3–6], only three instances are known in living individuals[7–9]. The first instance was reported in 1961 (ref. 7) from Prof. Böök's laboratory and occurred in a male infant aged 14 months. In common with the two subsequent cases, the infant was a chromosome mosaic containing both diploid and triploid cells, the latter having the suggested origin from a second fusion with a retained polar nucleus. The most recent observations on this child after an intervening period of three years have shown that the proportion of triploid cells in the skin tissues has markedly decreased from an original figure of 84 per cent to a value of 10 per cent; the decrease being credited (from observations on tissue cultures) to abundant mitotic irregularities in the nature of bridges, lagging chromosomes and tripolar spindles. It should, however, be pointed out that no mitotic instability has been observed in any of the other instances of human triploidy, and it remains to be seen whether the triploid cells in the two other living cases are selectively eliminated. It is further noteworthy that, in the case reported by Ellis *et al.*[8], the child was considerably older (6 years) and yet

* The Galton Lecture delivered before the Eugenics Society in London on May 20, 1964.